

# PEDIATRIC SURGERY

BY

EDWARD C. BRENNER, A.B., M.D., F.A.C.S.

DIRECTOR OF SURGERY, RIVER'S ISLAND HOSPITAL; DIRECTOR OF SURGERY, DETENTION  
HOSPITAL; ATTENDING SURGEON, MIDTOWN HOSPITAL; ASSOCIATE PROFESSOR OF  
CLINICAL SURGERY, NEW YORK POST GRADUATE MEDICAL SCHOOL, COLUMBIA  
UNIVERSITY; ASSOCIATE ATTENDING SURGEON AND CHIEF OF CLINIC, POST-  
GRADUATE HOSPITAL; CONSULTING SURGEON, HUNT'S POINT HOSPITAL;  
FELLOW OF THE AMERICAN MEDICAL ASSOCIATION; AMERICAN  
COLLEGE OF SURGEONS; NEW YORK ACADEMY OF MEDICINE;  
FORMER SURGEON, SQUADRON A.

ILLUSTRATED WITH 293 ENGRAVINGS



LEA & FEBIGER  
PHILADELPHIA

IN LOVING MEMORY OF  
OUR DAUGHTER

# PREFACE

THROUGH increasing knowledge of the affections of childhood and the judicious application of operative procedures suitable to the child's competency Pediatric Surgery has become an accepted branch of general surgery. Its importance has been emphasized for many years at the New York Post Graduate Medical School of Columbia University through both didactic and clinical teachings.

Students have often requested copies of the lectures and have expressed a desire for a text on the subject. It is purposed therefore to present a volume neither encyclopedic nor compendial in type which may be of practical value to those engaged in the study and practice of Pediatric Surgery. To this end illustrations have been freely employed and bibliographies and theoretical discussions omitted.

It is obviously impossible to review fully all the surgical diseases of childhood in a single volume. In order to allow sufficient space for the discussion of important and common pathologies it seemed advisable to omit fractures, dislocations and other orthopedic conditions since they are well considered in numerous textbooks.

Special attention has been directed to diagnoses, indications for operation, surgical therapy and the end results thereof. Conditions peculiar to infancy have received special emphasis and anatomy and embryology have been stressed only when necessary.

The child's body is no place for heroic surgery and the procedures recommended are based upon approved methods or those evolved from the writer's experience. Many operations are described in detail and the essentials of pre- and postoperative treatment are duly emphasized.

A well-chosen and skilfully administered anesthetic being of prime importance in the surgery of young patients a chapter has been devoted to the subject through the courtesy of Dr. T. Drysdale Buchanan.

Blood Transfusion has been written by Dr. Lester J. Unger whose experience in this special field is recognized.

In the presentation of *Congenital Cleft Lip and Palate* by Dr Harold S Vaughan the technic of each operative procedure is fully described and illustrated

Dr Louis R Davidson has discussed *Thoracic Surgery* from standards suitable to young patients

Many affections of the urologic tract require the care of a specialist Drs Clarence G Bandler and Arthur H Milbert have discussed these problems

The section on *Neurologic Surgery* has been contributed by Dr John E Scarff This timely presentation is necessarily abridged

To the foregoing the author expresses his sincerest thanks and appreciation

Gratitude is also extended to Drs William H Meyer and L Gregory Cole for the privilege of using certain x ray photographs and to Mr Karl K Bosse the artist for his excellent drawings

I am especially indebted to Elsie W McClellan my secretary who patiently aided in the preparation of the manuscript and meticulously read the proofs

Finally deep appreciation is due my sympathetic and understanding wife for her inspiration and comforting tolerance

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# PEDIATRIC SURGERY.

## PART I.

### GENERAL CONSIDERATIONS

#### CHAPTER I

#### INTRODUCTION

THE surgery of children is predicated upon the fundamental facts that infants and children differ from adults in anatomy, physiology and particularly in their reaction to operative trauma, and that the necessary adjustments of surgical procedures are not merely matters of scale. The immature organism is predominantly unstable and such incompetency forbids heroic surgery.

Conservative procedures, however, carefully planned and skilfully executed, are well borne even by infants. They do not tolerate well the loss of blood and meticulous hemostasis is imperative. They are also particularly sensitive to exposure and trauma and a prolonged operation or the rough handling of tissues may produce lethal shock.

The outcome of any surgical procedure depends greatly upon the child's state of nutrition and satisfactory water-saline balance. Conditions of starvation and dehydration with resultant acidosis or alkalosis should always receive appropriate preoperative and postoperative treatment through the administration of carbohydrates and saline in adequate amounts. Such combative and supportive measures are definitely life-saving and the value of repeated chemical blood examinations cannot be overstressed.

**Temperature and Respiration**—The average temperature in childhood is slightly higher than in adults and elevations and depressions are more readily produced by slighter causes. The respiration of infants is very irregular and the rate is subject to considerable variation. It averages 25 to 35 per minute during the first year and gradually diminishes to approximately 20 at five years. The character of breathing is chiefly abdominal until the tenth year, after which the costal type predominates.

**Pulse** The child's pulse tends normally to be somewhat irregular in character and frequency and the arrhythmia becomes increasingly more frequent from the age of six years to puberty. The pulse rate is highest at birth and gradually diminishes with age. During the early months it averages 130 to 140 beats per minute between the first and second years 110 to 120 and from two to four years 90 to 110 from which period it gradually lowers to 80 or 90 beats at puberty. Trifling causes as crying or excitement may increase the rate 20 to 30 beats.

**Blood Pressure** Before the third year it is impossible to determine blood pressure by the usual methods. From the ages of three to eight years the systolic pressure averages 80 to 90 mm. of mercury and the diastolic about 70. From then until puberty the systolic pressure rises to a maximum of 100 and the diastolic to 80 mm.

**Blood Volume** The blood volume of very young infants averages one-nineteenth of the body weight and at one year one thirteenth to one-tenth. The necessity for meticulous hemostasis during any surgical procedure cannot be overstressed; a loss of 10 per cent of the blood volume is serious and that of 20 per cent is generally lethal.

**Erythrocytes and Hemoglobin** The number of red blood cells and the percentage of hemoglobin are higher at birth than at any other time of life; during the first day the former may reach 6 000 000 or 7 000 000 or higher and the latter 150 per cent. A decrease in the erythrocytes occurs rapidly and a diminished number obtains until the eighth year when the adult standard is reached. The percentage of hemoglobin also falls rapidly and at six months averages 70 per cent. This gradually rises to the adult standard at puberty.

**Leukocytes**—In the first few days of life there is a leukocytosis of 16 000 to 20 000 which diminishes to 12 000 or 14 000 by the second week. Little change occurs during the first year but after this period the number approximates 9000 until the sixth year and 8000 thereafter. Whereas the polymorphonuclears may average 70 per cent at birth the lymphocytes predominate within a few days. Throughout infancy the proportion of lymphocytes and allied forms varies from 50 to 60 per cent and the polymorphoneutrophils approximate 40 per cent. This proportion gradually alters until the adult standards are reached at eight to ten years. The normal limit of neutrophils in early life should not exceed 60 per cent.

**Bleeding and Coagulation Time**—The bleeding time normally varies from one to three minutes but in the new born it may be prolonged. The coagulation time varies from three to nine minutes with an average of five. A delay of more than ten minutes is considered pathologic.

**Agglutinins and Receptors** Both may be present in the blood of even the youngest infant and in the selection of a donor for trans-

fusion the blood should be both typed and cross matched. Mothers appear to be safe donors for their new born without compatibility tests in only 87 per cent of case. (See Chapter VII.)

**Mortality** — Nutritional disturbances rather than surgical complications cause an alarming percentage of the surgical deaths in early life. Meticulous preoperative and postoperative care through close cooperation of the pediatrician, biochemist and surgeon is essential if the best interests of the child are to be served. Through such teamwork many apparently hopeless children may be salvaged.

**Diagnosis** — Diagnosis is especially difficult in early life because the intense constitutional reaction to disease often overshadows the focal symptoms. Moreover at this period pathologic processes progress with great rapidity.

Many diseases are not only peculiar to certain periods of growth but their manifestations vary when observed at different stages of development. Knowledge of the usual surgical conditions which arise at different ages is accordingly of great help in evaluating the symptomatology.

**In the New born** — Certain affections occur which may be due to developmental defects or to injuries or infections arising at birth or shortly thereafter. The former comprise malformations of the intestine and brain, congenital abnormalities visible to the eye and such inherited afflictions as syphilis. The latter includes meningeal hemorrhage and injury to the brain or nerves, dislocations and fractures, cephalhematoma and hemorrhage into the viscera and affections peculiar to early life such as pyloric stenosis, icterus and certain forms of sepsis.

**During the First Year** — Scurvy, rickets and affections of the thymus gland become evident at this period. Tuberculosis is also encountered usually as a generalized infection. Hyperplastic and tuberculous adenitis begin to appear and intussusception is definitely more common than at a later age.

**During the Second Year** — Diseases of the lymph nodes become especially common along with hypertrophy of the adenoids and tonsils. Tuberculosis is prone to be manifested focally as peritonitis, adenitis, meningitis or bronchopneumonia. In early childhood appendicitis begins to be observed, being rare before the age of three years, and with advancing childhood diseases of the bones and joints become more common.

As the organism becomes more stabilized the response to external stimuli is less intense and more focal. This tendency to the limitation of symptoms to a particular organ or region becomes most pronounced as the child approaches puberty. Whereas digestive disturbances commonly accompany many acute infections in early life the occurrence of nausea and vomiting in older children often denotes intestinal pathology.

**Convulsions** The significance of convulsive seizures also varies with the age of the child. Very young infants are comparatively free from convulsions and a seizure in the first or second months of life is almost always due to gross injury or infection of the central nervous system. From the third month to the completion of infancy the nerve centers attain their greatest irritability and convulsions are common and relatively inconsequential. Beyond the second year however they again become of ominous import.

## THE HISTORY AND EXAMINATION

Before examining the child a complete history should be elicited from the mother or nurse and the facts evaluated according to the intelligence of the narrator. Whereas factual statements from an observing mother are invaluable those from an excited parent are often misleading. Supplementary interrogation of older children is also of value and such sympathetic questioning of the patient prior to examination will frequently gain friendship and confidence. It is often surprising how an intractable child will become cooperative through recognition of its importance. The ego differ little from an adult's except that it is less restrained.

**Chronologic History** Except in emergent traumatic conditions it is best to proceed with the history in a methodical chronologic order. The age of children under two years is best expressed in months; in the new born the number of hours should be indicated. The latter is important in such conditions as imperforate anus, intracranial hemorrhage or fracture. The sex should also be recorded although aside from malformations of the genito-urinary organs the surgical diseases of both sexes are practically the same. Male children alone exhibit hemophilia.

Although race and nationality have little effect upon the incidence of surgical affections in childhood the prevalence of rickets among Negroes and Italians is worthy of mention. The family history should be elicited regarding hemophilia and in suspicious cases syphilis and tuberculosis. There may also be certain familial tendencies toward rheumatism, psychoses and tumor growths.

**Importance of the History**—At times the history is of more importance than the physical examination and the diagnosis of such conditions as acute appendicitis or intussusception can often be made over the telephone. The time of onset should be fixed as accurately as possible for the duration of the disease may not only influence the prognosis but also determine the advisability of emergent treatment. The necessity of immediate operation in acute appendicitis of twelve hours standing differs greatly from that of several days duration.

**Association of Trauma.**—In certain conditions the history of trauma should be carefully investigated. Cases of acute haematogenous osteomyelitis are frequently mistaken for rheumatism, and fracture of the infantile femur for scurvy or epiphysitis. The parent should also be questioned concerning the child's susceptibility to upper respiratory infections as the latter may influence the choice of anæsthetic.

**Vomiting.**—In cases of vomiting, careful inquiry should be made as to its frequency, the duration of the intervals between attacks, character of the vomitus, and whether projectile or regurgitant, also if there has been any provocative dietary indiscretion.

**In Suspected Intestinal Obstruction**—It is important to know not only when the last bowel movement occurred, but whether it was accompanied by blood and if so, was the latter mixed with the feces or with mucus, also if any flatus has since been expelled. The question of distention should be carefully investigated with reference to its development, duration, progress and the presence or absence of peristaltic waves. In giving enemias in such cases, the nurse should be warned not to admit any air with the fluid as its return may be mistaken for the passage of gas. Failure to pass flatus with repeated enemias definitely denotes obstruction.

**Pain**—The history of pain should be elicited in detail, the exact time and character of onset, its severity, location and radiation, and whether it has been of constant, remittent or intermittent type. Obstruction of the intestinal tract, whether it be of the appendix or gut, produces hyperperistalsis with resultant colicky pain referred to the periumbilical region. With the onset of peritoneal involvement, however, the pain becomes constant and focalized. In the former condition associated with paroxysmal pain, the patient is restless and tosses about whereas in the presence of peritoneal irritation he lies still, often with updrawn knees. The sudden cessation of severe abdominal cramps followed by focal tenderness in the right lower quadrant should strongly suspicion gangrenous appendicitis.

**Physical Examination.**—Although a complete physical examination is highly desirable, in many instances it is advantageous to proceed at once to the focal condition. A sympathetic approach will usually dispel the child's fears. Young infants are often best examined in the mother's arms, and nursing is at times a good decoy. In cases of older children, a gentle examination accompanied by tactful remarks inspires confidence and friendly cooperation. If the examination may cause pain, the child should be so informed. A trusting child will frequently exhibit great fortitude: even in the presence of peritonitis it may submit to rectal examination.

without reproach if the well-lubricated examining finger is slowly and gently inserted

Satisfactory examination of a highly neurotic or intractable child is at times impossible. In such cases a general anesthetic is advisable if an acute pathology is suspected. Either inhalation anesthesia or avertin by rectum may be elected. The intravenous administration of barbiturates is not recommended.

Biochemic, microscopic and bacteriologic findings are frequently of great diagnostic aid. In conditions of starvation and dehydration, estimation of the  $\text{CO}_2$  combining power and of the blood serum chlorides is especially valuable. Leukocytosis and polynucleosis in inflammatory conditions should be evaluated according to the child's age.

## CHAPTER II

### WOUNDS OF THE SOFT PARTS

**BRUISES** and minor lacerations are common in children. They generally heal kindly and require little care after the first dressing. Bruises may be relieved by the application of cold compresses or an ice-cap. Slight lacerations are best treated by cleansing with warm soap water and applying one half strength tincture of iodine acetone or 2 per cent mercurochrome. The last is least irritating and is preferable in young patients. The sterile gauze dressing may be kept in place by a bandage adhesive plaster or a covering of cotton and collodion. Wounds about the eyelids and lips are best treated with boric acid ointment.

**Early Wounds** — Gaping lacerations seen within the first twenty-four hours may be sutured or approximated with flamed adhesive plaster strips provided the wounds have been properly sterilized and all devitalized tissue has been debrided. The recognition of such tissue is aided by the application of 1 per cent aqueous methylene blue the dead tissue being stained more deeply. In the case of puncture wounds and those contaminated by soil or street dirt a prophylactic dose of 1500 units of tetanus antitoxin should be administered after determining the patient's sensitivity to horse serum. (See section on Tetanus.) When doubt exists as to the sterility of the wound a small rubber band or tissue drain should be inserted for forty-eight hours. The soft parts may be approximated with No. 0 plain catgut and the skin with dermal suture or horse hair.

**Late Wounds** — Wounds seen after twenty-four hours and all those which are grossly contaminated should be thoroughly cleansed, debrided if necessary, and lightly packed with gauze saturated with one half strength iodine solution. Extensive lacerations should be treated by the Carrel-Dakin method. Wound sterilization is best obtained in this manner with little discomfort to the patient. Secondary closure is performed when the results of smears and cultures from the wound surface are satisfactory.

**Lacerated Tendons and Nerves** — Primary tenorrhaphy and neurorrhaphy should always be attempted. Through delay the proximal ends of tendons retract and subsequent search for them may require an incision of considerable length. The Bunnell technic of tenorrhaphy is preferred by the author. (Fig. 1.) Following the

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suture of tendons or nerves on the palmar aspect of the hand or fingers the parts should be held in acute flexion by means of a molded plaster splint repairs of the extensor tendons require immobilization in hyperextension Following tenorrhaphy passive motion is begun on the tenth day and active motion on the fourteenth The results are generally very satisfactory The technic of neurorrhaphy is described in Chapter LII

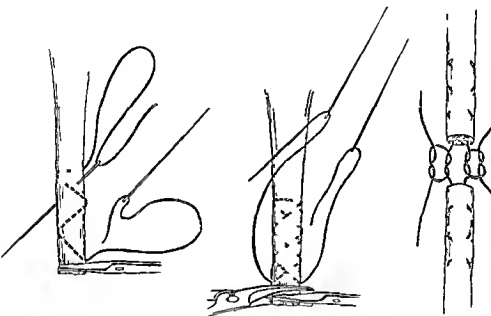


FIG. 1. Technic of tenorrhaphy. The tendon is held taut with a fine hemostat while the doubly threaded silk is passed into the tendon. Very little suture is exposed. Each damaged tip is cut off and the ends are approximated as illustrated in the lowest diagram.

### TETANUS (LOCKJAW)

Tetanus is an acute infectious disease which results from wound infection by the *Bacillus tetani*. The toxins produced thereby affect the central nervous system and the resulting increased reflex excitability and hypertonus cause persistent tonic contractions of one or more groups of voluntary muscle. Paroxysms of spasm aggravate the condition and in some instances focal paralysis results.

The tetanus bacillus discovered by Nicolaier (1884) is an anaerobic slender rod which grows in chains and is often enlarged at one end due to spore formation. The spores are among the most resistant known and have been found viable even after eighteen years. The normal habitat of the bacillus is in the intestinal tract

of herbivorous animals. Garden soil and street dust, contaminated by their excreta, are especially apt to contain the bacilli.<sup>1</sup>

**Incidence.**—This serious and entirely preventable complication of wounds occurs at all ages. It may develop in the new-born from infection of the umbilical stump and at times the puerperal mother is affected. Approximately one-half the cases occur in children between the ages of five and fifteen years. Rarely a case develops from spores in improperly sterilized catgut. Considering the wide distribution of the bacilli, the comparative infrequency of the disease would indicate that the organisms only proliferate in certain types of wounds and possibly only in those in which other organisms are present. Puncture and lacerated wounds, in which there is tissue destruction, are most favorable to tetanus infection. However, it may occasionally follow superficial lacerations of the skin or, rarely, of the mucous membranes. The bacillus neither causes suppuration nor prevents primary union and the wound may heal before the disease is manifested.

**Incubation Period.**—This varies from two days to eight weeks (Park), the average being about one week. The interval represents the time required for the passage of toxins from the site of injury to the spinal cord. In general, the earlier the onset the more virulent is the infection.

**Pathology.**—The bacillus is seldom found in distant tissues. It has been demonstrated occasionally in the lymphatics of nerves supplying the wound area but very rarely in the spinal cord. The pathogenicity of the organism depends entirely upon the development of soluble toxins in the wound: tetanin, tetano-toxin and spasmotoxin (Brieger). These appear to be absorbed by the end plates of the motor nerves and traverse the axis cylinder or possibly the perineural lymph spaces to reach the central nervous system. The irritant toxins produce hyperemia of the affected nerves and spinal cord, especially the cells of the anterior cornua. The hemorrhages found at times in the basilar meninges or brain tissue probably result from convulsions.

**Symptomatology.**—There is often the history of a splinter, rusty nail or blank-cartridge puncture injury. In some instances, however, the lesion is only a slight break in skin continuity. Thus, barefoot children occasionally develop the disease from trivial foot wounds which may have escaped notice and healed. Prodromal symptoms of headache, slight fever and soreness or stiffness in the vicinity of the wound may or may not occur. The first sign is generally spasticity of the muscles of mastication, especially the masseters, which causes difficulty in opening the mouth. Following

<sup>1</sup> Tetanus is more frequent in the Atlantic States than elsewhere in the United States

the trismus or lock jaw the cheek muscles may become tonically contracted and produce the expression of risus sardonicus.

**Progress of the Disease**—The muscles of the neck particularly the sterno-cleido mastoid and trapezius often become involved early followed by those of deglutition with resulting dysphagia. With further progress the back muscles and diaphragm may tonically contract and lastly those of the lower extremities producing opisthotonos. The order of involvement of the different muscles is quite variable. Clonic spasms are superimposed upon the constant tonic contractions and are often precipitated by trivial stimuli such as noises closing a door or jarring the bed. Separated at first by long intervals the clonic spasms become progressively more frequent and cause intense suffering. The pitiable victims remain mentally alert anxious and apprehensive. Occasionally certain groups of muscles become paralyzed.

The degree of fever is no index of the severity of infection. It is usually moderate but may rise suddenly in the terminal stages to  $107^{\circ}$  F. or higher. The pulse varies with the temperature and the respirations may become affected through involvement of the diaphragm. Emaciation is rapid. Death may result within a few days from respiratory paralysis glottic spasm cardiac failure exhaustion or aspiration pneumonia. Cases surviving the first week usually recover.

**Atypical Tetanus**—The foregoing account of a typical case offers no difficulty in diagnosis. However atypical manifestations of cephalic and of localized tetanus occur occasionally which require early recognition and vigorous treatment.

**Cephalic Tetanus**—This type follows head injuries and develops in two forms the non paralytic and paralytic. In the former trismus and spasms of the neck and pharyngeal muscles predominate the pharyngeal spasms may simulate those of hydrophobia. The diaphragm may also become involved. Most cases terminate fatally. The paralytic type develops from injuries to regions innervated by the trifacial nerve and the spasms of the cervical and facial muscles are associated with complete or incomplete facial paralysis. Oculomotor palsy may occur when the focal infection is about the eyes. The vocal cords and muscles supplied by the hypoglossal nerves are rarely involved. Many cases recover with complete restoration of function.

**Localized or Focal Tetanus**—This is an unusual manifestation in which tonic and clonic spasms occur in certain muscles near the wound but in which spreading or generalized convulsive seizures do not develop. Thus a single limb or group of muscles may be painfully involved in spasm. The prognosis is excellent. It is probable that many cases recover under erroneous diagnosis.

**Tetanus Neonatorum**—This form usually develops in the first week of life but has been reported as late as the fifteenth day. Although premontory irritability and sleeplessness may occur the first symptom is generally dysphagia—the infant attempts to nurse but cannot. The lips are drawn and the tongue is thrust forward. The progress of the disease is similar in all respects to that in older children. The mortality is exceedingly high and according to some observers approximates 90 per cent. This may be partly accounted for by lack of early diagnosis and timely treatment.

**Chronic Tetanus**—The term is applied to cases which develop after a long incubation period and pursue a chronic course. The same strain of symptoms may occur but they are generally modified in severity. Most cases recover.

**Diagnosis**—Whereas typical cases are easily diagnosed unusual forms may be confusing. In *strychnine poisoning* the repeated spasms are clonic in character followed by complete muscular relaxation. In *hydrophobia* there is a history of dog bite and the spasms occur chiefly in the muscles of respiration and deglutition. They cease entirely between attacks and the patient usually develops delirium. *Atypical meningitis* may simulate tetanus but is readily differentiated by lumbar puncture. *Tetany* is distinguished by the age in which it occurs and by the jerking character of the spasms which affect chiefly the hands and feet (carpo pedal spasm). The peculiar grouping of the involved muscles in tetanus neonatorum differentiates the pathology from that of *intracranial injuries*.

**Prognosis**—The mortality in the new born is exceedingly high perhaps 90 per cent. In older children about one-half succumb. Influencing factors are (1) a short incubation period which usually indicates severe infection (2) delayed diagnosis (3) belated serum treatment and (4) insufficient dosage of antitoxin. The occasional case which develops tetanus after prophylactic treatment usually pursues a mild course with recovery. Cases living beyond a week generally recover and the longer the patient survives the better the prognosis becomes. Paralyzed groups of muscles almost always regain function.

**Prophylaxis**—Tetanus prophylaxis is one of the outstanding triumphs of preventive medicine. In the United States Army during the World War only 36 cases of tetanus were recorded in 176 000 injuries. Prompt efficient sterilization of the wound is the most important factor in prophylaxis. This comprises mechanical cleansing disinfection with full strength tincture of iodine removal of foreign bodies débridement of devitalized tissues and adequate drainage. Routine administration of tetanus antitoxin is indicated in all lacerations which may be contaminated by garden soil manure or street dust and especially in puncture wounds from splinters rusty

nails blank cartridges compound fractures and burns. The serum should be administered promptly either intramuscularly or subcutaneously and preferably in the vicinity of the wound.

**Caution**—To safeguard against allergic reaction the patient should be questioned concerning asthma hay fever or any previous injection of horse serum. *Sensitization should always be determined by a preliminary subcutaneous injection of 0.1 cc. of the serum.* If no untoward reaction of erythema or severe itching is produced within ten minutes the full prophylactic dose may be administered with safety. Severe allergic reactions are rare occurring about once in 20 000 cases. In such instances 5 to 15 minims of 1 to 1000 adrenalin hydrochloride solution should be administered intramuscularly and be repeated if necessary.

**Prophylactic Dosage** For ordinary wounds 1500 units of antitoxin suffice if injected within twenty-four hours. When several days have elapsed 3000 units should be administered. In severely contused wounds contaminated with foreign material a second prophylactic dose of 1500 units should be given after one week as the immunity may not be effective after ten days. (Tetanus toxoid for the production of lasting immunization is still in the experimental stage and should not be employed in children.)

**Treatment**—Recent investigation indicates that antitoxin should be given in massive doses irrespective of the patient's weight. In the first twelve hours of the disease the initial dose should be at least 60 000 and preferably 90 000 units. This should be administered intrathecally intravenously and intramuscularly in doses of 20 000 to 30 000 units by each route. Another dose of 40 000 to 60 000 units should be given in a similar manner after twenty-four hours. The therapeutic value of intraspinal medication cannot be overemphasized and if it is instituted within six hours of the development of symptoms over 70 per cent recover. In favorable cases improvement occurs within forty-eight hours of the first injection. Should a relapse occur additional serum is indicated.

**Technic of Intrathecal Therapy**—The intraspinal injection of antitoxin should be administered slowly either by gravity or injection and preferably under an anæsthetic. The serum should be carefully warmed in a water bath to not above 100° F. as heat renders it inert. Puncture is performed between the third and fourth lumbar vertebrae and the cerebrospinal fluid is allowed to flow until its pressure falls to normal  $\pm$  1 drop every three to five seconds. The antitoxin is then allowed to flow in by gravity or is injected slowly allowing five minutes for the introduction of each cubic centimeter of serum. Subsequent injections may be more rapid. Should respiratory or other allergic manifestations occur 5 to 15 minims of 1 to 1000 adrenalin hydrochloride solution should be immediately injected intramuscularly. Cisternal puncture is only

indicated in cases of spinal block. The technic requires special experience.

If for any reason intraspinal injection cannot be administered 60 000 units should be given intravenously and 30 000 intramuscularly. When only a limited amount of antitoxin is available it should be given intraspinally instead of intravenously.

Neglected wounds may require exploration and subsequent disinfection. Healed ones however should not be reopened. The patient should be in a dark room free from all sounds if possible. Prolonged warm baths every four hours are soothing and may diminish the frequency of paroxysms. Maintenance of the normal water-salt balance is imperative. (Refer to Dehydration.)

**Sedation**—Convulsions may be controlled or ameliorated by chloral hydrate, bromides or barbiturates. In tetanus neonatorum the subcutaneous administration of 10 per cent solution of magnesium sulphate may relieve spasm and enable the infant to nurse (0.2 gm. of the salt is given for each kilogram of body weight).

## RABIES

### (HYDROPHOBIA)

Rabies is an acute specific infectious disease of the central nervous system communicated to humans by the saliva of a rabid animal usually a dog. It is characterized by a long incubation period, short clinical course and fatal termination. Approximately one half the cases occur in children.

Various strains of the virus apparently differ in infectivity as only about 35 per cent of humans bitten by a rabid animal develop hydrophobia if untreated. The nearer the bite is to the brain the greater is the likelihood of infection and bites upon exposed surfaces are more dangerous than those through clothes as the latter may filter the virus. The disease may also be transmitted by rabid saliva contacting cuts or abrasions or by its entering the conjunctival sac. Moreover the saliva may be infectious for as long as fifteen days before the animal manifests the disease.

**Incubation Period**—This generally varies from forty to sixty days and is rarely less than fourteen or more than ninety days. During this period the patient remains apparently well.

**Pathology**—Although the causative organism has not been discovered certain Negri bodies occurring in the larger cells of the central nervous system are considered morphologically specific. These should always be sought when the suspicious animal is autopsied. They are not always present however and when absent test animals should be inoculated.

**Symptomatology**—The prodromal symptoms of headache, malaise and slight fever are soon followed by those of nervous irrita-

bility and hypersensitiveness to light and drafts. In a few days the neck muscles become stiffened and there is difficulty in swallowing. Even the sight of water excites severe reflex spasms of the glottic neck and pharyngeal muscles (hence the name hydrophobia). The body musculature may later become involved in spasmodic contractions resembling tetanus. The attacks are intermittent however and the free periods are usually accompanied by delirium and hallucinations. Death frequently occurs suddenly from cardiac failure or the patient may pass into a paralytic stage and become comatose. Atypical paralytic or dumb rabie is a rare form of the disease in which progressive paralysis is the dominant syndrome.

**Diagnosis.** The condition of hysterical hydrophobia may be differentiated by the incubation period, lack of progressive symptomatology and susceptibility to suggestion. In tetanus the spasms are continuous and the muscles of mastication rather than those of deglutition are affected.

**Symptoms in a Rabid Dog.** Furious rabies is more common than the dumb type. The animal becomes irritable, wanders about, develops an abnormal appetite and snaps at objects and people. He fails to recognize his master or obey commands, scrapes continuously, has difficulty in swallowing and drops saliva (no mouth foaming). Spasmodic seizures soon develop and gradually pass into a stage of paralysis. Death occurs within four to eight days from the onset of symptoms. In the dumb type paralysis dominates the picture and the lower jaw is usually first involved.

**Treatment of Dog Bites.**—When the bite is upon the face or any exposed surface, immediate excision of the wound is the safest practice. When impractical, free bleeding should be induced by scrubbing the wound followed by cauterization with fuming nitric acid or the actual cautery. Primary suture in suspicious cases is particularly dangerous. Bites from healthy pets may be treated the same as any potentially infected laceration.

**Pasteur Treatment.**—The principle of the treatment discovered by Pasteur (1885) consists in the establishment of active immunity during the period of incubation by means of graded injection of an attenuated virus. The treatment is imperatively necessary when humans are bitten by animals known to be rabid. It is also indicated when the animal cannot be found or if it has been killed too early to develop clinical manifestations or exhibit Negri bodies. In such cases test animals should be inoculated. During this experimental interval treatment should be instituted for two weeks if the animals show no evidence of the disease. Should symptoms develop in the animals at a later period the treatment must be resumed immediately. Dogs which are apparently healthy should be kept under observation for at least three weeks and treatment



should be begun at the first sign of suspicious symptoms. Prophylactic treatment is also indicated if patients have been exposed to the saliva of rabid animals unless abrasions and cuts can be excluded. Infection is also possible through the conjunctival sac from saliva getting into the eyes. (When serving as an interne, the author was subjected to such infection from the saliva of a rabid police sergeant. The treatment was only locally disagreeable.)

Patients should be warned before taking treatment that the injections may produce local pain and erythema and in some instances febrile reactions. The mental solace, however, far outweighs the physical suffering.

Humans who develop hydrophobia are entitled to strong sedation as soon as the diagnosis is definitely established. Morphine and hyoscin should be administered in large dosages, also chloroform inhalations during the severe paroxysms. Restraint is often necessary when delirium develops.

## CHAPTER III

### BURNS AND SCALDS

BURNS may be thermal chemical electrical or result from radiant energy (radium and roentgen ray) Scalds represent a corresponding effect produced by hot liquids or steam Despite modern improvements in heating and lighting the incidence of burns shows little apparent decrease About one-third occur in children under ten years of age and no other type of injury causes such intense suffering high mortality or prolonged morbidity

Burns are more serious in children than in adults Their tissues are more delicate and greater damage results from lessened heat exposure Moreover the area involved is proportionately greater because of their small stature Burns covering one sixth of the body surface are often fatal and recovery is extremely rare when one-third is involved even though superficially

**Burns versus Scalds**—In burns the hairs are scorched or burnt off whereas in scalds they are unchanged but may fall out later Burns are usually more circumscribed than scalds and tissue destruction in the latter is generally superficial due to rapid cooling of the liquid or steam and its absorption by clothing The depth of protein coagulation depends upon the conductivity of the part involved and heat dosage *i. e.* the intensity of heat and its duration of contact Strong acids and alkalis produce a condition analogous to that of thermal burns and scalds

**Classification of Burns** Burns have been variously classified but for practical purposes the tissue damage may be superficial intermediate or deep the first second or third-degree types being characterized respectively by dermatitis blebs or eschars The clinical picture is generally a combination of variable degrees of tissue destruction and the severity of the lesion usually proves greater than is at first apparent This is especially so in electrical and radiant burns

**Shock**—This is inevitable in burns of any magnitude and the degree is influenced by the age of the patient situation of the burn and the extent of surface involvement The younger the child the greater is the relative shock a disproportionate degree may occur in young infants from even small burns Lesions of the trunk are more serious than those of the extremities and extensive surface burns cause greater pain and shock than circumscribed deeper penetrations

**Reaction in Serious Burns** — Patients extensively burned have little initial pain but complain of feeling cold and may have a chill. Grave cases generally become comatose and die within twenty-four hours from cerebral and visceral congestion. If reaction be established death may result later from profuse suppuration, septicæmia, secondary hemorrhage, gastro-intestinal inflammation with vomiting and bloody diarrhea or anuria.

In less serious burns a period of reaction follows the initial shock during which there is moderate elevation of pulse and temperature but the general condition appears satisfactory. Such improvement however may be deceptive. In a few days the temperature and pulse may rise, restlessness and delirium develop and the patient be overwhelmed by toxæmia. This is apparently due to the absorption of highly toxic products resulting from protein autolysis of the dead tissues. Somatic complications are not unusual: bronchopneumonia with chest burns, diarrhea with abdominal lesions and urinary suppression with flank involvement.

**Dehydration** — This is a serious factor in severe cases; an increasing erythrocytosis accompanies its development. (See Dehydration.) Acidosis may also be associated. Concentration of the blood results in circulatory insufficiency and subnormal tissue oxygenation. The latter favors the development of sepsis.

**Reaction in Favorable Cases** — In favorable cases there is a gradual subsidence of symptoms. First and second-degree burns heal spontaneously. In deeper lesions the destroyed tissues are gradually autolyzed and extruded and the raw surfaces become covered with granulations. Large defects often require skin grafting as epithelialization is slow and unsatisfactory. Moreover the resulting scars have little resistance and keloid formation frequently develops if infection occurs.

**Prognosis** — The prognosis depends chiefly upon the age of the patient and the extent and situation of the burn. The younger the child the graver is the prognosis. Burns of any magnitude in infants should always be considered potentially lethal. The surface area involved rather than the depth of tissue destruction is the more important factor. For this reason scalds which are apt to be superficial but extensive have a high mortality. Involvement of more than one-sixth of the body surface in infants or more than a third in older children is usually fatal irrespective of the depth of tissue destruction. Lesions of the trunk are graver than those of the extremities. Burns of the mouth, pharynx and epiglottis from inhalation of steam or hot gases are exceedingly dangerous. Persistent vomiting or serious irritability is ominous. Most deaths occur within the first week.

**Treatment** — The potential lethal factors are shock, toxæmia and sepsis. These require both constitutional and local treatment.

Pain and shock are best alleviated by hypodermic injections of codein in infants and morphin in older children repeated prn. The foot of the bed should be elevated and the body temperature maintained by local heat in the form of hot water bottles electric pads or the electric light cradle. Hot retention enemias of 10 per cent glucose solution may be administered every two hours. Immersion in a continuous bath of 5 per cent sodium bicarbonate solution kept at a temperature of 100° F. is excellent for small children. The treatment may be continued for several days until the sloughs have separated and granulations appear.

To prevent the development of anhydremia and acidosis abundant fluids should be administered in the form of water sweetened drinks and fruit juices supplemented when necessary by proctoclises hypodermoclises or phlebotomies of 3 per cent glucose in physiologic saline solution. (See Dehydration.) There is an apparent analogy between the toxemia of burns and that of high intestinal obstruction in that tissue retention of sodium chloride occurs in both. Saline clises are therefore specifically indicated.

**Burns and Scalds of the First degree** — Measures should be directed toward the relief of pain the prevention of infection and the preservation of function. Wet dressings of 10 per cent bicarbonate of soda solution allay pain and have a mild antiseptic value. A perforated rubber tube inserted in the gauze mesh permits frequent injections to keep the dressing moist. (For first aid at home baking soda may be used.) The burned area should be moved frequently and contiguous joints flexed and extended. In facial burns gum chewing is helpful. The sodium bicarbonate dressings are washed off daily with sterile water and after the third day the parts are exposed to sunlight for ten minutes before the dressings are reapplied.

Following subsidence of the dermatitis boric acid ointment or sterile vaseline may be employed to hasten desquamation. Between dressings the burned area should be exposed to increasing doses of sun rays or ultra violet irradiation. (The rays from unfrosted electric light bulbs may be substituted.) Burns of this variety leave no scar.

**Paraffin Treatment** — This has many advocates. The burn area is covered with a layer of warm paraffin applied with cotton or a brush. (Melted candle shavings may be substituted.) Over this is placed a  $\frac{1}{4}$  inch layer of sterile gauze. Warm paraffin is then poured over the dressing and another layer of gauze applied. The dressings are readily peeled off in forty eight hours and may be renewed or followed by boric acid ointment or sterile vaseline applications.

**Severe Second and Third-degree Burns or Scalds** — Pain and shock demand primary attention. The clothing should be carefully removed or cut away to prevent injury to blebs and vesicles. These wounds are potentially infected and require sterilization. One-half

strength tincture of iodine may be applied to small burns and 1 per cent picric acid to larger ones. Continuous wet dressings of 10 per cent sodium bicarbonate solution or the paraffin treatment may be employed. Blebs should not be opened until the third or fourth day when, after sterilization with iodine the base of the sac may be aseptically punctured and the serum allowed to escape. The epithelium should not be removed as it serves as a sterile protection.

*Warm Air Treatment*—After subsidence of the acute dermatitis (two to four days) warm air treatment is beneficial. The patient is placed in the nude beneath a cradle in which a few electric light bulbs are strung. The cradle is covered with sterile sheets and the temperature within is maintained at 100° to 105° F. The lights both ray the burned area and keep the patient comfortably warm. This form of treatment is especially well borne by infants and small children. If crusts form or the treatment produces discomfort, dressings saturated with equal parts of sterile olive and camphorated oils may be applied. The crusts afford a protective covering and should not be removed unless pus accumulates beneath them. When the healing stage is reached open air treatment with graduated exposures of sunlight hastens repair.

*Tannic Acid Treatment*—E. C. Davidson has made a noteworthy contribution to the treatment of burns in advocating the use of tannic acid. The chemical tans and coagulates the protein tissue and tends to prevent absorption of the toxins which result from the autolysis of dead tissue. The tannic acid may be applied on compresses saturated with a 2 to 4 per cent freshly prepared aqueous solution. (Tannic acid in solution is unstable and changes to gallic acid.) A coagulum is rapidly produced thereby. When exposed to the air, it forms a brown parchment-like covering which is non-sensitive and prevents loss of body fluids. Tannic acid ointment, 5 per cent, is also recommended. For rapid tanning, the patient should be placed under an electric light cradle and the burned areas sprayed every thirty minutes with 4 per cent tannic acid solution from an atomizer. (The eyes should be protected.) Complete tanning may be obtained in twelve to eighteen hours.

Blebs should be opened early and their contents evacuated to permit deeper penetration of the tannic acid. In superficial burns, epithelization proceeds beneath the coagulum. Crusts are not disturbed unless pus accumulates beneath them. They usually fall off in ten to fourteen days, leaving a healed surface. In deeper burns, the coagulum adheres to the charred tissues and requires removal in about two weeks. The partially liquefied dead tissue is cut away with it and the wound allowed to granulate in preparation for skin grafting. Dakinization is valuable at this stage. If the entire circumference of a limb is involved, vertical incision should be made in the tanned tissue to aid circulation.

*Advantages of Tannic Acid Treatment*—Tannic acid treatment definitely lessens toxemia aids in preventing infection and conserves the loss of body fluids. Its other advantages are ease of application, simplicity of after care and the comfort afforded the patient. The author recently had under care a child of four years with second degree scalds involving the lower abdomen, groins and



FIG. 2.—Clean burn wounds of the buttocks following 4 per cent tannic acid treatment

both thighs. The patient was placed under an electric light cradle and the scalds sprayed with 4 per cent tannic acid every thirty minutes for sixteen hours. Opiates were discontinued the second day. The highest temperature was  $101.4^{\circ}\text{F}$  and at no time was there evidence of toxemia or infection. Thiersch grafts were applied on the nineteenth day. The child was discharged in six weeks with wound healing complete and function unimpaired.

**Burns About the Eyes** — These are usually treated with wet compresses of 2 per cent boric acid solution. In cases of conjunctivitis 1 per cent argyrol may be instilled tid. With subsidence of the acute dermatitis the parts may be exposed to the air with graduated sunlight exposures.

**Débridement** — Small penetrating burns and those containing foreign material from explosions may be débrided. If this be done early immediate skin grafting may be attempted. Many surgeons prefer grafting at a later date when healthy granulations have developed and the smears indicate a relative sterility (not more than two organisms per field).

All burns are potentially infected wounds and suppuration may develop irrespective of any type of treatment. *B. pyocyaneus*, *staphylococcus* and *streptococcus* are the usual organisms. With a rise of temperature all crusts and blebs should be carefully examined for infection. Greenish blue *pyocyaneus* pus is inconsequential it may prevent mixed infection and apparently does not retard healing. If maggots appear in the wounds they should be undisturbed. They ingest bacteria and dead tissue and thus induce healthy granulations. (Their artificial introduction is not recommended.) Wound sterilization is best obtained by the Carrel Dakin technic of intermittent hypochlorite irrigation. Cases of prolonged suppuration are greatly benefited by repeated blood transfusions. If the same donor is reemployed compatibility should be redetermined. (Refer to Chapter VII.)

**Electric Burns** — These usually result from contracting high voltage circuits. They are heat burns differing in no way from those produced by other high temperatures and require similar treatment.

**Radiant and Roentgen ray Burns** — Degenerative processes result from absorption of the irritating rays. The injury is insidious produces no immediate discomfort and the lesion does not usually appear for several days. Superficial burns produce dermatitis with loss of hair. The skin may atrophy later from destruction of the nutrient vessels and telangiectases often develop in the scar. In deeper burns the degeneration is more penetrating. After remaining healed for several years crusts warts keratoses and fissures may develop in the skin and undergo epitheliomatous degeneration. Radiant burns are very resistant to treatment. Small types are best excised and the defect covered with full thickness skin grafts.

**Contractures** — Burns about the neck axillae groins flexor and extensor surfaces of the joints and the hands often result in serious limitation of function through cicatricial contractures. (Fig. 3.) Prophylaxis is most important and whatever form of treatment is adopted preservation of function becomes imperative. This is best accomplished by early motion passive or active and by appropriate

posture. In burns about the face, gum-chewing or blowing of balloons should be prescribed early. In lesions about the anterior surface of the neck, the head should be held in hyperextension and lateral motion encouraged. When the axilla or groin is involved the arm or leg should be kept in abduction (Fig 3). In burns about the extensor or flexor surfaces of joints the parts should repose in flexion or extension respectively. Joint motion should be maintained from the start. Manual extension of a limb is prefer-



FIG 3 —First-stage plastic repair of a preventable axillary contracture (Courtesy of Dr J J Moorhead)

able to constant traction. Deep burns of the palmar surface of the hands are often best treated by debridement and full thickness pedicle grafting of the raw surface.

### KELOIDS

Keloids occur often in burn scars, especially following infection. Developing as flat red shining hard tissue elevated above the skin they may be sensitive, at times cause itching and frequently produce contractures which limit function. Histologically keloids



consist of dense fibrous tissue in which there is much collagenous material, separated by a few cells and blood-vessels. The extension of the cells along the vessels into surrounding tissues may account for the recurrences following surgical extirpation.

**Etiology.**—The etiology of keloid formation is little understood. The chief factors appear to be infection and a fibroplastic diathesis. The latter occurs most often in negroes, is more frequent in brunettes than in blondes and is seldom present in albinos. Susceptibility may be acquired in some instances as is illustrated by the following case: Appendectomy was performed when the child was four years of age and healing occurred with a hair-line scar. At seven years a second-degree scald of the back was sustained and several large keloids developed in the scars. When twenty years of age, the patient was operated upon by the author for hygroma colli. The wound healed *per primum* but developed an ugly keloid within three months. Such acquired susceptibility may last throughout life.

**Treatment.**—None is definitely satisfactory. Small keloids may soften and shrink during the course of several months. The implantation of a few strands of paraffined silk, acting as setons, may hasten shrinkage. Excision with the scalpel or endotherm knife is uncertain and may result in a larger secondary keloid. The Morestin method of central oval excisions, in multiple stages, is less apt to produce recurrence but necessitates repeated operations. The most satisfactory methods of treatment appear to be (1) complete excision of the keloid and approximation of the normal skin edges without tension, followed by radiation eight or ten days postoperatively, and (2) excision of all scar tissue and whole thickness skin grafting of the defect.

## CHAPTER IV

### SURGICAL SHOCK DEHYDRATION ACIDOSIS ALKALOSIS

#### SURGICAL SHOCK

SURGICAL shock is a condition of profound depression of all the vital functions of the organism. Although trauma is practically always the exciting cause conditions such as hemorrhage sepsis starvation and undue exposure to cold may augment its degree. Functional depression of the vasomotor center from mental disturbance may produce a similar condition termed collapse.

Capillary stasis is the dominant factor in the physiology of shock and the diminished volume of blood in the vital circulation causes a marked fall in arterial blood pressure. Insufficient tissue oxidation resulting therefrom reduces the alkaline reserve and a state of acidosis thus becomes superimposed.

**Symptomatology**—The patient is profoundly prostrated and lies still and listless. The features are drawn the eyes sunken and the skin pallid slightly cyanotic and clammy. The pulse is rapid, often almost imperceptible at the wrist, and the blood pressure is extremely low. The respirations are shallow sighing and irregular. Except in extreme cases the condition tends to recovery and after a few hours the circulation may become stabilized the respirations normal body warmth and color return and the sensorium brighten. A gradual rise in blood pressure is often the first favorable sign prolonged low blood pressure is ominous. In unfavorable cases all the symptoms become exaggerated and the patient lapses into unconsciousness and succumbs. Infants and young children are more susceptible to shock than adults but they all react more promptly to treatment.

Hemorrhage is often associated with shock and the clinical picture may be altered by the symptoms of restlessness hunger and thirst. The presence of leukocytosis strongly suggests internal bleeding hemoglobin and erythrocyte estimations are less reliable. A fall in blood pressure occurs in both conditions and at times it is very difficult to evaluate the relative importance of the various factors.

**Treatment**—Preoperative prophylactic measures are extremely valuable in minimizing the likelihood of the development of shock. A normal water saline balance with adequate carbohydrate fortifi-

cation is highly important (See chapter on Preoperative Treatment) Cathartics are ill advised. The anesthetic should be chosen carefully and administered skilfully. Prolonged surgical procedures, rough handling of tissues and undue loss of body heat through exposure should be particularly avoided. Bleeding is especially dangerous and meticulous hemostasis should be maintained at all times. Repeated observations of the blood pressure should be recorded during the operation. In cases of falling systolic pressure a supportive infusion of 10 per cent glucose in physiologic saline solution is indicated.

When shock is threatened or apparent the patient should be placed in the recumbent position with the foot of the bed elevated 45 degrees and be surrounded with warm blankets and hot water bottles supplemented by an electric light cradle. In severe shock the transfusion of whole blood is the most valuable known agent. When impractical saline infusion of 5 to 10 per cent glucose may be substituted. In mild cases the hypodermochysis of 3 per cent glucose saline solution usually suffices. At times a hot retention enema is also helpful. Stimulation with camphor in oil, caffeine or brandy although often employed appears to be of little benefit. Rest is imperative and is best secured by small doses of codein or a barbiturate.

## DEHYDRATION OR ANHYDREMIA

Adequate water balance is necessary to life and when the amount of fluid intake plus that produced by metabolism is less than the quantity eliminated dehydration results.

To guard against physiologic withdrawals of fluid an available reserve of loosely bound water is provided in the skin, subcutaneous tissues, muscles and liver. This may be generously depleted without producing symptoms or change in the water content of the blood as is commonly evidenced by profuse sweating after vigorous exercise. When the withdrawal is too severe however the blood shows increased concentration and symptoms of anhydremia develop. A loss of 10 per cent of the body fluid produces serious symptoms and 18 to 20 per cent is generally lethal.

**Etiology**—Excessive loss of water occurs most commonly in repeated vomiting and in severe diarrhea, also in hyperpyrexia, hemorrhage, burns, extensive discharging wounds, peritonitis and starvation. The water reserve in infants and young children is relatively small and early recognition of tissue desiccation is therefore extremely important.

**Symptomatology**—The symptoms are largely the result of decreased blood volume. One of the earliest is rapid and excessive loss of body weight. The features become sharpened, the eyes

sunken and the skin dries and loses its elasticity. The latter may be demonstrated by pinching normally a fold of pinched skin returns promptly to its previous level whereas in dehydration it is putty like and disappears slowly.

The patient breathes with open mouth and the tongue and lips become parched dark red and cracked. The pulse is small at times irregular and there may be slight fever. The urine is scanty, highly concentrated and frequently contains albumin and casts. The blood may reveal mild erythrocytosis and leukocytosis, an increase of non protein nitrogen and a decrease of the bicarbonate content. Acidosis is a frequent complication due to lactic acid accumulation and the retention of anions.

**Treatment**—This comprises removal of the cause when possible and prompt restoration of the normal water balance. Except in mild cases water by mouth is inadequate. Proctoclysis is messy and impractical in young children and the required amount of fluid should be administered by hypodermoclysis, phlebotomy or intraperitoneal injection. In the average case hypodermoclyses of 3 per cent glucose in isotonic saline solution are administered every four to six hours in amounts of 80 to 300 cc. depending upon body weight. The glucose acts as a readily oxidizable food aid in combating acidosis and promotes diuresis. Some clinicians favor the intraperitoneal injection of isotonic glucose (6 per cent) in physiologic saline solution. (Abdominal distention occurs less often if the glucose is dry sterilized before being dissolved.)

**Blood Transfusion**—Severe destruction of the red blood cells occurs in prolonged dehydration and blood transfusion may be necessary to enable the body to assimilate the injected fluids. It is definitely indicated when the hemoglobin falls below 60 per cent. The change brought about by the transfusion of whole blood followed by repeated hypodermoclyses of physiologic salt solution is often phenomenal.

In extreme cases of dehydration an infusion of 1 per cent glucose solution may be administered *after* each saline clasis. The dosage is 10 cc. of 10 per cent glucose solution for each pound of body weight. This occasionally aids in restoring water balance in an otherwise hopeless case.

### ACIDOSIS

The term acidosis denotes a condition in which there is a relative increase of acid in proportion to the normal alkali in the blood plasma, body fluids and tissues. Although the alkaline reserve is reduced a true acid state never occurs.

**Discussion**—The acid base balance of the body is usually expressed in terms of the pH and  $\text{CO}_2$  combining power of the blood plasma. Although the pH purports to evaluate the reaction of the

body fluids it is frequently perplexing to the clinician. The  $\text{CO}_2$  C P which may be designated as the alkaline reserve or  $\text{CO}_2$  capacity, is a means of reporting the amount of sodium available for combination with  $\text{CO}_2$  released in the tissues.

$\text{CO}_2$  occurs in the blood stream in two forms, carbonic acid and sodium bicarbonate. Normally there is approximately 20 times as much of the alkali as of the acid and so long as this ratio is maintained the pH remains at the normal level.

Variations in the relative amounts of bicarbonate and carbonic acid affect the pH level, the volume rising with an increase in the alkaline factor and falling with an increase in the acid. In all conditions involving a primary change in bicarbonate concentration the pH shifts in the same direction; i. e. if the base is depleted by accumulation of acids from incomplete combustion of fats, the pH is lowered, indicating an abnormal relative acidity.

Effort is made to prevent the pH from varying beyond the normal limits (7.34 to 7.45) through altering the rate of  $\text{CO}_2$  removal from the lungs. When the carbonic acid concentration can be raised or lowered to compensate for the bicarbonate change, the pH will remain normal and the condition may be described as compensated acidosis or compensated alkalosis, according to the shift in the bicarbonate.

The determination of the sodium bicarbonate content of the blood plasma is commonly reported as the  $\text{CO}_2$  combining power. This varies normally from 50 to 70 volumes per cent on ordinary oxalated blood. Values below this level denote varying grades of acidosis, whereas concentrations in excess of the normal signify alkalosis.

With  $\text{CO}_2$  C P values between 40 and 50 volumes per cent a slight acidosis exists. Due to the conditions under which the test is ordinarily made, it is best to regard 40 to 50 volumes per cent as low normal. Such findings are very common in hospitalized patients. Determinations from small children frequently fall within this range without evidence of an actual acidosis. It is possible that the alkaline reserve of children is not so great as in adults, or that the drop in bicarbonate is induced by the apprehension attending the collection of the blood. In our experience (Mattice) an alkalosis has been strongly suspected in children when the  $\text{CO}_2$  C P has exceeded 60 volumes per cent.

For practical purposes it is unnecessary to estimate the pH of the blood as determination of the alkaline reserve (the  $\text{CO}_2$  C P) provides the clinician with all the information needed with reference to the acid base balance.

**Etiology**—Carbohydrate starvation is the commonest factor in the production of acidosis. Incomplete fat combustion resulting therefrom causes the retention of acetone bodies. Dehydration, which is often associated, also favors acidosis through lactic acid

accumulation and the retention of anions. Repeated emesis occurring in peritonitis and in intestinal obstruction may produce profound acidosis. Severe diarrhea is also a common causative factor.

The disturbance of the acid base balance associated with general anesthesia may be regarded as an acidosis. (1) the  $\text{CO}_2\text{CP}$  is diminished. (2) there is retention of  $\text{CO}_2$  as  $\text{H}_2\text{CO}_3$ , and (3) the pH is depressed. The anesthetic interferes with normal oxidative processes leading to ketogenesis, accumulation of lactic acid and to release of phosphoric acid. Bridges determined the alkaline reserve pre- and postoperatively in a large number of children subjected to tonsillectomy. In all cases the  $\text{CO}_2\text{CP}$  was diminished 10 volumes or more one hour following the operation as contrasted with the control samples taken immediately before the anesthetic.

**Symptomatology**—The younger the child the greater is the predisposition to the development of acidosis. Certain symptoms are suggestive: the cheeks are often flushed, the lips ruddy and the tongue beefy red. There is frequently an early restlessness and sleeplessness which may gradually pass into somnolence and coma. In most instances the breath has an acetone odor (simulating that of chloroform). The most significant symptom of severe acidosis however is hyperpnea, although the respiratory rate may not increase, the inspirations and expirations become exaggerated and prolonged.

**Diagnosis**—Positive urinary findings of acetone, diacetic and beta-oxibutyric acids are conclusive evidence of acidosis. Their absence however does not exclude the condition as acidosis may rarely occur without ketosis. *The ultimate diagnosis depends upon the presence of an abnormal decrease in the carbon dioxide combining power of the blood.*

**Preoperative Prophylaxis**—Since carbohydrate starvation and dehydration are the important factors in producing acidosis, preoperative fortification is highly desirable. The glycogen reserve in the liver and muscles should be increased by giving meals rich in carbohydrates to within six to eight hours of operation. In older children hard candy may be added. Fluids and fruit juices should also be administered in abundance and all fats omitted. Vigorous catharsis should be avoided; an enema may be given the night before or morning of operation. The choice of anesthetic and its skilful administration also assume an important role. (Refer to Chapter VI.)

**Treatment of Impending Acidosis**—When acidosis is imminent or present isotonic glucose (6 per cent) in physiologic saline solution should be administered either by infusion or intraperitoneal injection. The dosage varies from 80 to 300 cc. depending upon body weight. If the intraperitoneal method be employed the glucose should be dry sterilized before being dissolved in sterile distilled water. Distention is generally prevented thereby. The transfusion

of whole blood is beneficial when the hemoglobin is below 65 per cent

**Postoperative Acidosis**—Prompt restoration and maintenance of the normal water saline and acid base balance is imperative in post operative acidosis. When the normal fluid intake is insufficient it should be supplemented by the administration of physiologic saline solution through hypodermoclysis, pleuroclysis or intraperitoneal injection. Isotonic glucose should be added for hypodermoclyses, however the content should not exceed 3 per cent. In diabetic patients the same procedure is followed adding 1 unit of insulin to the solution for each 3 gm. of glucose.

The therapeutic value of alkali administered in the form of sodium bicarbonate is uncertain and unsatisfactory. Frequent  $\text{CO}_2$  blood estimations should be made if large doses are prescribed as excessive alkalinization has been known to produce a lethal alkalosis. (Nurses should be specifically warned against boiling sodium bicarbonate solutions as the alkali may be reduced to highly toxic sodium carbonate. Deaths have resulted therefrom.)

## ALKALOSIS

Alkalosis is a condition in which there is an abnormal increase in the alkaline reserve of the body cells, intercellular fluids and blood plasma. In some instances it may follow the excessive intake of alkali. The normal kidney is capable of excreting sodium bicarbonate in a concentration of approximately 15 gm. per liter. If the kidneys are incompetent or an overdose of alkali is administered accumulation may result and the alkaline reserve become elevated. A similar condition may ensue when the electrolyte content of the body fluids is reduced through the loss of  $\text{HCl}$  as in persistent vomiting.

The clinical conditions which commonly induce alkalosis are pyloric stenosis, cyclic vomiting, gastric dilatation, high intestinal obstruction and occasionally pyelitis. It may also result from hyperventilation in hysteria, postencephalitis, hyperpyrexia and anoxemia. The urine is low in fixed bases and the excreted acids are either free or bound to ammonia. Ketone bodies may also be present.

**Diagnosis**—As in acidosis there are no pathognomonic symptoms. Nausea, vomiting and especially numbness are suggestive. In severe cases tetany and convulsions may occur. Diminution or absence of chlorides in the urine should arouse suspicion. *The accurate diagnosis depends upon the laboratory finding of an abnormal increase in the  $\text{CO}_2$  combining power of the blood.*

**Treatment**—Removal of the cause is imperative. In cases due to an excessive intake of alkali, usually sodium bicarbonate, the medication must be withdrawn. Infusions or hypodermoclyses of

physiologic saline solution should be administered every four to six hours, in amounts varying from 80 to 350 cc., depending upon body-weight. One to 5 minims of 0.2 per cent hydrochloric acid may be added to the saline, or given by mouth. Its value, however, is questionable.

Pyloric and high intestinal obstruction demand prompt surgical intervention. A preoperative saline infusion, or blood transfusion, is advisable. At the completion of operation, the peritoneal cavity may be filled with isotonic glucose (6 per cent) in saline solution. Postoperatively, large amounts of fluid, preferably physiologic salt solution with glucose, should be administered rectally, intravenously or subcutaneously. (The glucose should not exceed 3 per cent in hypodermoclyses.) The sugar is readily oxidized, serves as food, and promotes diuresis.

Reduction of the hydrogen-ion concentration follows the administration of  $\text{CO}_2$  and the acid-producing salts, ammonium and calcium chloride; also, possibly, of dilute hydrochloric acid. If tetany develops, the most effective agents are ultra-violet irradiation and the intravenous administration of calcium chloride; acids and viosterol may be helpful adjuvants. Further knowledge of the etiology, prophylaxis and treatment of alkalosis is desired.



## CHAPTER V

### PREOPERATIVE AND POSTOPERATIVE REGIMEN

**Fasting** during the acute operative period comprising the day of operation and the two thereafter the tendency of the modern surgeon is to maintain food ingestion at a level approximating the normal. A regimen of preoperative starvation and catharsis is specifically interdicted. The former may produce definite alteration in the physiologic balance of the body fluids with resulting acidosis and the latter cause dehydration through disturbance of the normal water saline balance.

**State of Nutrition**—In cases of election it is important to have the child in the best possible state of nutrition before the surgical procedure is undertaken. Furthermore an excess of carbohydrates should be administered for some days prior to operation in order to combat the withdrawal of nourishment during the active operative period. This additional intake may be in the form of jams, honey, stewed fruits, sweetened drinks, hard candies or lolly pops. The omission of fats is specifically indicated.

**Carbohydrate Fortification**—The value of carbohydrate fortification in the prevention of post anesthetic acidosis is well exemplified by the experiments of Hawks. Dogs fed 3 to 4 gm. of carbohydrate per kilo of body weight failed to exhibit acidosis following anesthesia. When carbohydrates were withdrawn for ten days the same dogs showed constant post anesthetic acidosis.

The child should enter the hospital the day before operation and be kept at rest. Supper should consist of a cooked cereal well covered with sugar and an abundant intake of fluids. Laxatives are omitted. A warm soap-water enema is given in the morning and water is allowed until two hours prior to operation. In infants the last bottle or breast feeding may be given at midnight.

**Chemical Blood Study**—Cases suffering from serious surgical pathologies should have a preoperative chemical blood study to determine the CO combining power, blood sugar and chlorides. The nutritional state of the patient may be evaluated therefrom and appropriate combative measures instituted when indicated.

**Acute Operative Period**—The carbohydrate and fluid intake should be carefully maintained during the active operative period. When necessary supplementary glucose in physiologic saline solution may be administered either by hypodermoclysis or phlebotomy. Proctoclysis is messy and generally unsatisfactory in children. The fluid intake should equal or exceed the normal and in no case be less than 50 cc. per kilo of body weight during each twenty-four hours.

**Administration of Carbohydrates**—Fat and protein storage in the body is sufficient for several days providing an adequate water

and carbohydrate intake is maintained (Dogs have been kept alive experimentally or weeks solely through the parenteral administration of glucose in normal salt solution) Carbohydrates may be administered orally in weak tea or such sweetened beverages as lemon or orange phosphate and subcutaneously or intravenously in the form of glucose in physiologic saline solution The amount of glucose commonly administered in phlebotomies is 5 per cent in hypodermoclyses however it should not exceed 3 per cent

**Postoperative Feeding** Water is often permitted in small amounts as soon as the patient has recovered from the anesthetic Sips of hot water are tolerated better than cold cracked ice and iced drinks provoke colic On the second postoperative day fluids are given freely especially fruit juices On the third and fourth days milk toast cereals custards and ice cream are added and the normal dietary is rapidly resumed

**Nausea and Vomiting**—Postoperative nausea and vomiting are definitely lessened and frequently obviated by preoperative carbohydrate fortification The symptoms are generally inconsequential and subside within four to six hours Excessive vomiting however is serious and dehydration with resulting acidosis or alkalosis may result therefrom Although the vomiting is usually of toxic origin other causes such as gastric dilatation intestinal obstruction and dehiscence should be excluded A saline or soda bicarbonate autolavage (20 gm. of soda bicarbonate to the liter) will often relieve the condition When unsuccessful gastric lavage should be employed through the aid of a Levine tube introduced intranasally Nothing should be given orally until the vomiting has ceased for at least six hours

**Diet in Special Conditions** **Anemia**—In elective surgical conditions secondary anemia is best treated by a hygienic-dietetic regimen A high caloric diet rich in iron copper and manganese is supplemented with viosterol fish liver oils or their concentrates also hematinics heliotherapy and forced rest A natural increase in the blood elements is more lasting than the relief afforded by blood transfusion In emergent conditions however the latter is specifically indicated when the hemoglobin is below 60 per cent

**Underweight Children**—Malnutrition is largely due either to a chronic state of dehydration or to hygienic-dietetic errors The former should be combated by an abundant fluid salt and carbohydrate intake The latter requires a full balanced diet supplemented when necessary with fish liver oils and ample rest It is noteworthy however that postoperative metabolic disturbances develop less frequently in thin than in obese children

**Nephritis**—Severe renal damage interdicts surgery except when imperative In elective cases operation should be deferred until renal competency has become safely stabilized through the aid of diet An adequate preoperative carbohydrate and fluid reserve is

especially important as toxins are eliminated less promptly in the presence of renal insufficiency

**Diabetes Mellitus**—Since the advent of insulin, the dictum that "diabetes in early life knows no mercy" is no longer tenable. The following facts have been definitely established

1. In the absence of acute infection, sugar tolerance is not influenced

2. If the diabetes is under control, a constitutionally competent patient may be given approximately the same prognosis as the non-diabetic.

3. Clean wounds heal promptly and convalescence is not protracted

Although elective operations may be performed with relative safety, the writer believes that surgery should not be advised for the diabetic child except through necessity. Combative precautions are imperatively indicated. For a few days prior to operation the carbohydrate intake should be increased and if necessary the insulin as well, so that glycogen storage in the liver and muscle cells may be at its maximum

The choice of anesthetic agent is extremely important. Chloroform should never be employed. Ether is also contraindicated. blood sugar is increased, acidosis is favored through diminution of blood oxidation, the excretion of acetone bodies is diminished by the decreased urinary output, and the effect of insulin upon carbohydrate metabolism is largely neutralized while the organism is saturated with ether. (Refer to Chapter VI)

**Acute Appendicitis in the Diabetic Child**—This brooks no delay for perforation is attended by a much higher mortality than in the non-diabetic. In the presence of peritonitis, operation is imperatively indicated as the death-rate from the Ochsner method of treatment is prohibitive. Waiting to control the hyperglycemia and acidosis is a serious mistake for bacteria grow with astounding prodigality in peritoneal exudates containing a high-sugar content. Furthermore, the value of insulin may be decreased more than 50 per cent in the presence of infection and its efficiency only returns when drainage is established. During the acute postoperative period of supportive treatment, frequent determinations of the blood sugar, pH and carbon dioxide combining power are necessary in order to estimate the required insulin dosage

## CHAPTER VI

### ANESTHESIA

By T. DRYSDALE BUCHANAN, M.D.

THE reactions to various general anesthetic agents and the principles governing their administration are much the same with children and adults. However certain modifications as to dose and methods must be adopted if young patients are to receive the full measure of pain relief commensurate with safety.

**Physical Examination**—When arranging for an operation the same careful physical examination should be recorded the child as is given the adult including urinary blood and biochemic findings. *Abnormalities of glands and any deformities* that may interfere with unrestricted respiration should be especially noted. The anesthetist should be provided with all the data in order that the optimum agent and method of administration may be carefully selected.

**Carbohydrate Fortification** It should be emphasized that following ether or chloroform narcosis children are more prone to acidosis than adults also that the normal alkaline balance is less promptly restored. The preoperative diet should therefore be high in carbohydrates and on the eve before operation a cooked cereal plentifully covered with sugar should be given.

**Acidosis** If acidosis is present elective surgery should be postponed until a normal balance obtains. This is usually accomplished through the ingestion of carbohydrates and the addition of mineral water containing carbonates. When the operation is imperative acidosis is best combated through the intravenous administration of 5 per cent glucose in physiologic saline solution. (Refer to Acidosis.) The return of the normal alkaline state of the blood is deterred by the use of ether or chloroform. Nitrous oxide ethylene or cyclopropane diluted by oxygen and administered by an expert are definitely preferable since these agents are cleared from the blood stream and tissues in a few minutes.

**Dehydration.**—The prevention or correction of dehydration in children is even more important than in adults. A small retention enema of tap water may be given preoperatively or a supportive infusion of saline and glucose may be administered slowly throughout the operation the restlessness of children making this measure difficult to institute postoperatively. (Refer to Dehydration.)

**Preoperative Sedation**—Predicated upon clinical observation rather than scientific research data it is generally accepted that

children do not well withstand prolonged operations or lengthy anaesthesia. Nevertheless many children are thoughtlessly submitted to surgery without the administration of any prophylactic against shock although this need is greater than in adults. Avertin or a barbiturate may be prescribed with or without morphine.

**Avertin** — The administration of a tribromethanol such as avertin has robbed the approach to anaesthesia of most of its terrors. Such sedation should be utilized more frequently for children with their higher oxygen metabolism tolerate these drugs in basal doses even better than adults. The procedure is especially indicated when repeated anaesthetics are necessary as in multiple operations for skin grafting or painful osteomyelitis dressings.

**Barbiturates** — The judicious use of small doses of a barbiturate such as pentobarbital introduced rectally in capsule form one hour before the anaesthetic is started has proved to be an excellent procedure. It effects a mild form of basal anaesthesia or at least dulls the child's intellect so that psychic shock is minimized. The usual dosage given rectally is as follows: Up to four years of age 1 grain from four to eight years  $1\frac{1}{2}$  grains and from eight to twelve years 2 grains. Absorption will be more rapid if a needle puncture is made in each end of the capsule.

**Opium Derivatives** — These too often omitted in childhood can also be utilized to great advantage in avoiding many of the disagreeable incidents of inducing anaesthesia. They not only contribute to smooth narcosis throughout the operation but minimize postoperative discomfort. The practice of giving small doses of morphine in conjunction with pentobarbital has been followed for a number of years at the Hospital for Sick Babies in Toronto with most gratifying results. The following doses for full weight children not too weakened by pathology have been employed by Charles H. Robson M.D. anaesthetist to that institution.

Age	Morphine Sulphate	Pentobarbital
4-4½ years	$\frac{1}{4}$ grain	$\frac{1}{2}$ grain
6	$\frac{1}{4}$ "	$\frac{1}{2}$ "
8	$\frac{1}{4}$ "	1
10	$\frac{1}{4}$ "	1
12	$\frac{1}{4}$ "	$1\frac{1}{2}$ grains
	<b>Codeine Sulphate</b>	
9 years	$\frac{1}{4}$ grain	None
1 year	$\frac{1}{4}$ "	None

Modern respirators and the use of carbon dioxide and oxygen stimulation of the respiratory center have greatly lessened the hazard of morphine medication. When the drug is given preoperatively strict attention should be paid to the time of injection for the respiratory depressant action should be wearing off before the action of the anaesthetic is superinduced.

The experiments conducted by Ralph M. Waters definitely indicate that the acme of morphine action is reached in the majority of cases in one and a half hours. Accordingly the drug should be so administered that this interval may elapse before anesthesia is induced.

**The Problem of Approach**—This is of great importance. The anesthetist should inspire sympathy and confidence in order that fright and nervousness be minimized. Experience has taught that we are apt to underestimate the child's mental capacity and curiosity, thus tempting both the doctor and parent to tell a white lie about a picture being taken or an examination being made. It should be borne in mind that at some future time the patient may have to undergo another operation and lost faith is difficult to regain.

It is astonishing how often children will submit calmly to anesthesia if the phenomenon is explained to them beforehand in simple words and cheerful tones. When too young to understand recourse should be made to divert their attention by means of a toy, perfume on the mask, or some nonsensical conversation which delights them.

**Parental Cooperation**—The question frequently arises as to the advisability of allowing a parent to be with the child during the induction. This can only be determined in each individual case but more good than harm comes from this practice provided the parent is instructed in what to expect and agrees to leave when requested. Some rebellious children will only agree to begin the narcosis if a parent is allowed to hold their hand.

It is enlightening at times to witness the ingenuity with which children will delay the administration, often with the cooperation of the parent. One frequently witnesses ruses and tricks worthy of the brain of an adult and the anesthetist will be well repaid by tolerating these defensive antics. At times the patient can be fooled into drowsiness by passing a current of one of the gases over the nose and mouth.

In some instances, however, persuasion and patience are useless, rendering it necessary to hasten the administration by a rapid action induction agent as ethyl chloride dropped on an open mask, or nitrous oxide, ethylene or cyclopropane gas. This may appear brutal to the parent but needless to say, cutting short the fright period of struggling is the more humane method.

**Choice of Anesthetic**—The selection of the anesthetic agent and its method of administration will depend on the physical findings, the psychic make up of the child, the manner of elimination of the agent, and the type of operation to be performed. Of the numerous available anesthetics those most frequently employed are ether, nitrous oxide, chloroform and ethyl chloride.

**Ether** — At the expense of being thought elemental a short review of the action of these agents is presented. Ether a highly inflammable agent with a strong pungent odor is a cardiac and respiratory stimulant in non toxic doses. It produces irritation and congestion of all mucous membranes congestion of the brain an increased flow of saliva and acidosis. The initial rise in blood pressure caused by ether is sustained over a long period unless shock or severe hemorrhage occurs.

In the event of overdosage the respiratory center is overpowered before the cardiac. For this reason the drug has a wider margin of safety than any other anesthetic thus far produced. Although its use is generally contraindicated in severe nephritis bronchitis pneumonia and pulmonary tuberculosis the irritating effects on the organs involved can be somewhat modified by warming the vapors or through administering it with olive oil by colonic injection as first recommended by James T. Gwathmey, M.D.

**Nitrous Oxide** — This gas is odorless rapid in action and non irritating to the mucous membranes. It causes an initial rise in blood pressure and stimulation of the respiratory center. The latter is followed in overdosage by prolonged and difficult exhalation in direct proportion to oxygen want. The resulting cyanosis produces engorgement of the soft palate uvula and tongue which may lead to occlusion of the respiratory tract. It is contraindicated in young children and in all cases in which the pathology impairs free access to the lungs such as peritonsillar abscess Ludwig's angina and trismus from any source.

**Ethylene** — The agent is somewhat similar to nitrous oxide except that it has a slightly disagreeable odor acts more profoundly requires a greater admixture of air or oxygen and does not produce such harsh breathing. It is highly explosive and care must be taken to avoid static sparks canter points or actual flames as it is very easily ignited. Under proper conditions it is a safer anesthetic for children than nitrous oxide.

**Chloroform** — Chloroform is a respiratory and cardiac depressant and causes a fall in blood pressure with transient anemia of all organs and membranes. Since in high concentration or in liquid contact with the skin and mucous membranes it may produce burns or blistering the exposed parts should be covered with vaseline. It is non inflammable but decomposes readily in the presence of sunlight or actual flame. The chlorine liberated is extremely irritating to the respiratory tract. This decomposition will also occur in a closed room in which the available oxygen is consumed through overcrowding lighted gas jets or a grate fire.

Long chloroform administrations or repeated narcoses at short intervals may give rise to acute yellow atrophy of the liver and fatty degeneration of the tissues. Although the incidence has been

materially lowered through vaporizing the agent by means of a current of oxygen its use has nevertheless been largely abandoned.

In overdosage the cardiac and respiratory centers are supposed to be overcome simultaneously from anemia. Although recent investigators indicate that the respiratory center failure is followed very quickly by cessation of the heart action it is nevertheless true that fibrillation of the heart occurs if chloroform is pushed too rapidly or in too great a concentration. Its usage is definitely contraindicated in shock, anemia, obesity, myocarditis, low urinary output and suspected status lymphaticus.

**Ethyl Chloride** This anesthetic is extremely volatile and produces an immediate fall in blood pressure followed by rapid stertorous respiration and muscular relaxation. The drug has little to recommend its use in children except as a preliminary to ether. It is definitely contraindicated in cases with restricted breathing notably pneumonia, empyema and pneumothorax.

**Administration of the Anesthetic** In order that the patient be not kept waiting the anesthetist should be certain that the apparatus is clean and in perfect working condition, that a sterile hypodermic syringe, mouth gag, tongue forceps and small blade laryngoscope are at hand, that the oral, nasal and endotracheal airways are immediately available and that the necessary respiratory and circulatory stimulants including oxygen are within instant reach. A good respirator is an additional safety factor if the anesthetist lacks experience in resuscitation.

Even the bravest or most gullible little patient will be frightened if the mask is placed tightly on the face. Direct contact should not be made until the anesthetic has stupefied the patient sufficiently to obliterate all memory of the procedure. Patience is its own reward when narcotizing children.

Encouragement to count aloud with a cadence set by the anesthetist not only diverts the child's mind but is an excellent guide in governing the rapidity of induction. This diversion can be further helped by telling stories suitable to the child's mentality.

Following slow induction the anesthetic is pushed as rapidly as safety permits to the depth desired for the particular operation care being exercised to avoid cyanosis. The latter is absolutely unjustifiable; it is not only a constant menace to the surgeon but also damages tissues during its existence. Patients are not necessarily profoundly narcotized because they are cyanosed.

Continuous administration of ether by oral and nasal inhalation suffices for most operations. In cases requiring intraoral or intra-nasal procedures better results are obtained by pharyngeal or endotracheal insufflation or inhalation.

**Artificial Airways**—Whenever it is necessary to establish an artificial airway either for conveying the anesthetic vapor or for



resuscitative procedures the first choice should be the nasal passages. Children frequently have loose teeth which may become detached and aspirated into the trachea or bronchi through attempts to insert an oral airway.

The nasal airway is established as follows: two lengths of rubber tubing beveled at one end and cut long enough to extend from the nares to the level of the epiglottis are passed through the nostrils well down in the pharynx the outer ends being fastened together by a suture or safety pin. Greasing the tubes facilitates their introduction and avoids excess trauma.

**Endotracheal Anesthesia**—Certain cranial, spinal and thoracic operations demand proficiency in the art of introducing the anesthetic through the trachea or directly into one of the bronchi. In the occasional pneumonectomy, lobectomy or the repair of diaphragmatic hernia the operative risk is greatly minimized if instant inflation and oxygen supply to the lungs are made possible. This can only be vouchsafed by a constant access to the bronchi.

By means of specially curved catheters introduced by Dr. E. I. Magill of London it is possible to rapidly intubate the trachea through either nostril. With the patient in the supine position and the head in the median line a catheter with the largest caliber the nostril will tolerate is well lubricated and gently introduced to the level of the epiglottis. At this point some resistance is met but by waiting for the next inspiratory effort the tube can be readily slipped between the vocal cords and the intubation accomplished. The larynx must be sufficiently anesthetized either by general or local agents to abolish spasm. Coughing or short apnea often accompanies the intubation.

Absolute surety of introduction however requires visualization of the vocal cords through the aid of the laryngoscope. This requires considerable practice in order that it may be done with the minimum of trauma. Laryngitis of short duration is a frequent sequel even though tubes of molded rubber are used. *metal tubes increase this complication*

To and fro inspiration can be utilized by attaching the outer end of the tube to any suitable gas machine or ether may be dropped on a mask placed over the ingress of the tube and inhalation anesthesia be carried on. When lung inflation is required an insufflation apparatus is necessary.

*Endotracheal anesthesia when established is the safest known method of narcosis.* It provides ready access to the lungs for air or oxygen and maintains at all times an unobstructed passage for inflation or deflation of the lungs. Although some added risk is involved in the introduction of the catheter it is greatly minimized through skilful laryngoscopy.

**Endovenous Anesthesia** — In some clinics the endovenous method of introducing a brief acting barbiturate is employed for short operations upon older children. This procedure falls under the heading of irreversible anesthesia as do spinal injections and rectal ether. Spinal anesthesia and regional bloc are impractical in young patients.

In the event of an overdose or abnormal result from endovenous anesthesia one is dependent entirely upon the hypodermic or intravenous introduction of heart and respiratory stimulants and of minute doses of picrotoxin. Injections of the latter in 0.003 gm doses should be repeated as necessary. Stimulation of the respiratory center should also be augmented by inhalations of 7 per cent CO in oxygen or the use of the mechanical respirator.

An overdose of picrotoxin produces twitchings and convulsions resembling those of strychnine poisoning and should these occur they must be met by a return to the barbiturates. Picrotoxin is the only known detoxicant of the barbiturates. Cardiac and respiratory stimulants merely tide the patient over the depression of these centers.

**Open Drop Ether Anesthesia** This is unquestionably the safest and most satisfactory anesthetic for short operations as circumcision, myringotomy, incision and drainage, pyloroplasty, etc. For longer procedures the mask should be modified to allow for rebreathing so that too great a loss of CO<sub>2</sub> will not occur with resultant apnea or shock. A rebreathing chamber may be improved by building a towel tent over the mask leaving an opening at the top to admit sufficient air to avoid cyanosis.

If one employs ethyl chloride or chloroform by the open drop method the towel tent should be omitted and the mask held far enough from the face to allow a free current of air to circulate through. Otherwise the vapors may become too concentrated and prove lethal. Before inducing anesthesia the cheeks, nose and lips should be coated with vaseline to avoid blistering.

Vaporizing the chloroform by means of a current of oxygen directed through the liquid and carried by tubing to the mask is preferable to the drop method. It insures regular dosage, a more even plane of anesthesia and an adequate supply of oxygen throughout the narcosis. However there is little if any need for chloroform in the surgery of children.

**Dangers of Ethyl Chloride** Ethyl chloride is employed chiefly to precede ether when rapid induction is desired. The best and safest results are obtained by the open method with the mask held 1 inch from the face. Children respond to it very easily and pass from one stage to another so rapidly that it is only applicable to short operations. For longer procedures ether should be substituted as soon as rapid respirations and unconsciousness occur. Any

degree of fixed dilated pupil or shallow respiration is a warning not to be disregarded. Although experts are able to skilfully administer nitrous oxide ethylene or cyclopropane to young children so that satisfactory results are obtained the tyro or occasional anesthetist should not attempt to do this.

**Ethylene and Cyclopropane**—Older children are better subjects for ethylene or cyclopropane with oxygen providing a proper apparatus is used. Such an inhaler should provide for a constant flow of the gases accurately measured either in liters per hour or cubic centimeters per minute instantly changeable at the discretion of the administrator. A face piece that can be made air tight an attachment for measured dropping of ether a rebreathing bag and a chamber containing soda lime for absorbing excess  $\text{CO}_2$  are all essential if anesthetic is to be maintained on physiologic principles.

**Soda Lime Technic**—This is based upon the assumption advanced by Dr. Howard Haggard that inhalation anesthetics undergo no change in the body. Consequently once the proper anesthesia vapor tension in the blood stream is obtained the same plane of narcosis can be sustained by rebreathing the vapors without renewing the agent except for small amounts lost through leakage. Prolonged rebreathing however would result in a high concentration of  $\text{CO}$  in the rebreathing bag with consequent overstimulation of the respiratory center. To obviate this the expired air is directed through a soda lime chamber where most of the excess  $\text{CO}$  is absorbed. The cyanosis resulting from constant rebreathing is offset by a continuous flow of oxygen of 150 to 300 cc per minute.

The advantages of the  $\text{CO}_2$  absorption technic are manifold rapid and easy induction control of the respiratory rate with absence of harsh or forcible breathing avoidance of oxygen want an even plane of anesthesia at any depth desired a minimum of sweating a more rapid recovery with less nausea and lastly economy. The method has proved so satisfactory that it is being adopted throughout the country and many foreign anesthetists are visiting America for the purpose of learning its details of administration.

**Importance of Posture**—The best posture for most operations is the supine with the shoulders and thighs slightly raised to avoid abdominal tension and the head turned to either side. If the head is held in the median line relaxation will permit the lower jaw and base of the tongue to drop backward. Free action of the epiglottis is thereby inhibited causing varying degrees of respiratory obstruction.

**Tonsillectomy** is usually performed in the supine position with the head slightly extended over the end of the table or over a sand bag. Some surgeons prefer that the patient be strapped in a chair in a sitting position.

**Kidney operations** are usually done with the patient on one side raised in a jack knife position to widen the space between the ribs

and pelvis. Roentgen-rays have shown that this posture materially reduces the alveolar absorption space, and when the patient lies on the left side the heart is displaced to the right, rendering breathing more difficult. Patients should not be kept in this position longer than is necessary.

During the operations for *cleft palate* a free airway is easier of maintenance if the head is well extended over the edge of the table in the Rose position. The anesthetic may be insufflated through the nose or by means of a bent metal tube hooked at the outer border of the mouth.

*Removal of impacted teeth* may be performed under endotracheal or endopharyngeal insufflation, either method allows for packing off the upper part of the pharynx with gauze. If the pharyngeal method is chosen, the nasal airways can be attached to a Y metal tube through which air, oxygen and ether vapor may be delivered in the lower pharynx below the gauze packing.

In *cranial, mastoid and facio-plastic surgery*, the sterile field may be preserved by carrying the anesthetic to the patient through insufflation by either the pharyngeal method or the endotracheal route, preferably the latter.

*Anesthesia Following Hemorrhage*—Cases deprived of oxygen carriers through severe hemorrhage react better under an anesthetic agent that permits of a high-oxygen dilution such as ethylene or cyclopropane. Either may be supplemented with small amounts of ether if necessary.

Cyclopropane should be preceded by several inhalations of pure oxygen and then allowed to flow at the rate of from 300 to 600 cc per minute until anesthesia is manifested, when it is discontinued. Oxygen is then continued at the rate of 150 to 300 cc per minute until the operation is completed.

Although some doubt exists as to the advisability of carrying patients for over sixty minutes on cyclopropane, the agent is of great value in selected cases. A fixed eyeball or irregularity of the pulse, slow pulse, apnea or cyanosis are definite signs that the gas should be diluted with oxygen or stopped entirely. It should be noted that cyclopropane can be exploded and the same precautions must be used as in ether or ethylene administrations.

*Labored Breathing*—The gospel of safe anesthesia lies in a clear air passage at all times and nothing is more futile than artificial respiration in the presence of obstruction. Therefore, when breathing difficulties arise the first step is to establish ready access to the lungs. If the obstruction is due to a relaxed tongue impeding the epiglottis, free breathing may be established through pushing forward the lower jaw, changing the position of the head, wrinkling the skin of the neck toward the chin, or by inserting a nasal or pharyngeal airway.

If the cause of labored breathing is an accumulation of thick mucus in the nasopharynx, the throat should be cleared by means of suction. If vomiting occurs, the mouth and pharynx should be cleared before resuming the anesthetic.

At times the anesthetic is too light and reflexes cause cessation of respiration. This occurs most commonly when traction is made on the tonsil, mesentery, etc. The operation should be immediately stopped and if the apnea persists, a cross-reflex should be inaugurated through some procedure as slapping the chest with a wet towel, spraying the body with ether, stretching the anus, or alternate traction and relaxation of the tongue. When respiration is resumed the anesthesia should be deepened to avoid a recurrence.

An overdose of the anesthetic usually requires nothing more than withdrawing the anesthetic and substituting pure air or oxygen if the patient is still breathing. If the respiratory center is entirely depressed, artificial respiration by the Schneffer or Howard method is called for, together with endotracheal intubation so that the lungs may be inflated and deflated at will.

**Resuscitation**—Modern respirators are very helpful in maintaining prolonged efforts at resuscitation, and the use of inhalations of 5 or 7 per cent CO in oxygen is advisable. Respiratory stimulants such as alpha lobeline, coramine ciba or metrazol introduced intravenously, have produced good results, and the old method of mouth-to-mouth breathing is still considered an excellent procedure by many anesthetists. Infusions of saline and glucose are also beneficial.

**Cardio Puncture**—Heart stimulants for cardiac failure should be given hypodermically, endovenously or at times by direct injection in the heart muscle. In performing cardio puncture the needle should be inserted in an interspace hugging the right border of the sternum in order to reach the auricle, punctures on the left side of the sternum invariably reach the ventricular walls and produce ventricular fibrillation.

**Cardiac Massage**—Indirect cardiac massage may be accomplished by placing the two thumbs over the apex of the heart and vigorously punching down at the rate of 60 to 70 times per minute. If the abdomen is open direct massage may be performed by grasping the heart through the diaphragm and squeezing it at the same cadence. As a last resort, an intercostal incision followed by the insertion of a finger to allow pinching the heart against the chest wall has been recommended.

**Heart Failure**—The heart action generally persists for a certain period after the respiration has failed and one should be able to resuscitate the majority of respiratory failures. Once the heart has actually stopped, however, the chances of recovery are practically nil. Although the heart action may appear to have entirely

ceased, the electrocardiogram will at times reveal evidence of activities imperceptible to the finger or stethoscope. This is the type of case which is occasionally reported as having been brought back to life through injections of adrenalin or electrical stimulation of the heart muscle.

**Anesthesia in Special Conditions —Cardiac Disease —**Although cardiac pathology presents an added hazard, the risk is rarely sufficient to proscribe operations of necessity. Special care is required in cases of active carditis, and in the presence of rheumatic infection with accompanying muscle damage as evidenced by exercise tolerance.

Decompensated cases will not withstand prolonged strain or oxygen want. It is important therefore, to avoid or minimize any struggling during the induction of narcosis. This is best accomplished by preoperative sedation through the administration of avertin or pentobarbital by rectum. These agents do not affect the heart and should be given one hour before operation. Anesthesia is induced by open-drop ether given slowly until the patient is unconscious when the ether should be vaporized by oxygen. An unrestricted airway should be established promptly because cardiac strain is in direct proportion to respiratory strain.

It is imperative that such patients be watched constantly until recovery from the anesthetic is complete so that any respiratory obstruction which develops may be immediately corrected.

**Diabetes —**Diabetes is best combated through an added carbohydrate intake for several days prior to operation, neutralized by adequate insulin administration. Since ether and chloroform inhibit the action of insulin the last dose of the drug should be given two hours before inducing the narcosis to prevent its being antidoted by the anesthetic.

**Intestinal Obstruction —**In the presence of intestinal obstruction a preoperative gastric lavage should always be given to prevent the possible aspiration of toxic vomitus. This may be difficult to perform unless a previous basal dose of tribromethanol has been administered rectally. From 60 to 70 mg. per kilo should suffice.

**Status Lymphaticus —**Interval surgery is interdicted in the presence of an enlarged thymus gland or other symptoms suggesting status lymphaticus. When immediate operation is imperative, ether administered by the open-drop method is the safest agent, clinical experience has shown that nitrous oxide, ethylene, ethyl chloride, chloroform and cyclopropene are all in the doubtful column. It is far better to sacrifice the niceties of induction in the interests of a safe recovery.

The foregoing has been presented in the hope that the busy surgeon will find some useful hints of practical value in the surgery of young patients. It is in no sense a complete treatise on the

## CHAPTER VII

### BLOOD TRANSFUSION

By ILLSTIR J. UNGER M. A. M. D.

BLOOD transfusion has had a very varied career ranging from extreme popularity to the point where at one time in France it was actually prohibited by law. It is an extremely old procedure. The history of its development and the solving of problems which from time to time seemed insurmountable comprise an interesting chapter in the progress of surgery.

As the technic was improved and more and more transfusions were performed it soon became apparent that the mere passage of blood from donor to recipient caused death in many instances. Step by step the problem of incompatibility was solved and the technic of administration perfected. As a result, the transfusion of whole blood is now a safe and valuable therapeutic measure and nowhere is its popularity more noticeable than in the realm of pediatrics.

The indications for blood transfusion may be divided as follows:

- 1 Hemorrhage
- 2 Diseases of the blood
- 3 Infections
- 4 Toxemia
- 5 Shock
- 6 Miscellaneous conditions

**1 Hemorrhage**—Severe loss of blood is a most obvious indication for blood transfusion and the therapeutic results are more startling than those obtained in any other condition. Through replacement of the blood elements and control of the bleeding many otherwise hopeless cases are saved. The hemorrhage may be acute or chronic in nature. In the former the onset is sudden, the duration comparatively short, and the amount of blood that is lost is relatively large. The group includes such conditions as bleeding from the stomach or intestine, postoperative and traumatic hemorrhage.

When considering transfusion for an acute hemorrhage there are three facts which must be kept in mind:

(a) *The Dose*—At one time it was thought that it was necessary to estimate more or less accurately the amount of blood that was lost and to replace approximately that amount. We know now

that this is not so. In general a large transfusion is always advisable. It is unwise to determine any definite dosage beforehand and the amount given should depend upon the patient's condition at the time of the transfusion. The patient's interest will be better served by giving an amount which improves his general condition, evidenced by a slowing and better quality of the pulse, improvement in color and elimination of the cold clammy moist condition of the skin. In children the amount varies roughly from 10 to 15 cc per pound body weight. Although in comparison with the average adult dose this seems high it nevertheless gives the best results.

(b) *The Blood pressure* The question is often asked: Will the transfusion elevate the blood pressure so as to increase the hemorrhage rather than control it? The answer is that blood transfusion almost never increases the blood pressure more than 5 mm. of mercury except where there has been a marked antecedent fall. Even then however transfusion will not raise the pressure above what was normal for the patient. The procedure can be and should be employed with perfect safety even though active bleeding is taking place. Any hesitation on the part of the operator in this type case is due to fear rather than to any adverse experience. As a matter of fact a fall in blood pressure follows transfusion in some instances; a slight rise may be due to excitement.

(c) The third problem which presents itself is in connection with cases where an operation is contemplated. *Should the operation be performed first or should the transfusion take precedence?* The correct answer is contrary to the obvious one. The transfusion should precede the operation except in cases where an abdominal operation is to be performed upon a ruptured viscus. Assuming there is external bleeding immediate transfusion is indicated in order to combat shock and enable the patient to withstand the operative procedure. Although some of the transfused blood may be lost and a second transfusion become necessary the chances of recovery are much increased. In cases of hemorrhage from a ruptured spleen or kidney transfusion and operation should be performed simultaneously. As soon as the pedicle of the viscus is secured and hemorrhage thereby controlled the transfusion is started. An effort should be made to have the transfusion last during the entire period of the operation or at least until the surgeon begins closure of the wound.

*When the Hemorrhage Is of the Chronic Type*—In this type the onset of bleeding is more or less insidious, the hemorrhages are repeated and the amount lost at any one time is relatively small. The condition occurs most commonly in tuberculosis, ulcerative colitis, polypus coli and rectal polyp. Although blood transfusion is less effective than in acute hemorrhage it is nevertheless of definite value in improving the patient's condition through an increase of erythrocytes and hemoglobin.



**2 Diseases of the Blood**—Included under this heading are the following

- (a) Secondary anemia
- (b) Primary anemia
- (c) Hemophilia and purpura hemorrhagica
- (d) Leukemia
- (e) Bleeding in the new born
- (f) Rare blood diseases

(a) **Secondary Anemia**—Transfusion is extremely useful and the primary cause of the anemia is often itself improved or cured as a result. Where the primary cause is not removable as in chronic nephritis or chronic endocarditis blood transfusion should be employed as a symptomatic form of treatment.

(b) **Primary Anemia**—Transfusion is definitely indicated in the rare types of primary anemia in children.

(c) **Hemophilia**—This form of blood dyscrasia transmitted through the female to the male is frequently confused with purpura hemorrhagica. The two diseases may be readily differentiated by certain laboratory tests. In hemophilia the coagulation time is prolonged while in purpura hemorrhagica it is normal. There are many methods by which the coagulation time may be determined. Most of them however are more or less inaccurate the degree of accuracy varying with the method chosen. Any technic which obtains blood from the capillaries through skin puncture is subject to error because the blood is invariably mixed with a certain amount of tissue juice which diminishes the coagulation time. It is therefore advisable to discard the first few drops of blood but even then a certain amount of tissue juice is carried with the specimen.

*The Coagulation Time*—Of the various methods for determining the coagulation time among the simplest are the capillary pipette technic and that of Biffi Brooks. Blood is usually obtained by puncturing the finger or the lobe of the ear. The first few drops are discarded and the blood is then allowed to fill the fine pipette by capillary attraction. Every one-half minute a piece of the tube containing the blood is broken off and when the blood in the fragment is coagulated the end point of the reaction has been reached. The difference in time between obtaining the blood and that of its clotting is the coagulation time. The norm by this method is four to six minutes.

The Biffi Brooks technic is somewhat more accurate because it utilizes an apparatus which takes into consideration the temperature of the air surrounding the specimen of blood. Cold lengthens the coagulation time and by having a more or less constant temperature one variant is eliminated. Moreover the drop of blood to be tested is held in a fine platinum loop so that it contacts a minimum amount of foreign material. The two factors tend toward greater

accuracy. The normal coagulation time by this method is five to seven minutes.

In the test tube method blood is withdrawn from a vein as for a Wassermann test. After the first few cubic centimeters have been discarded approximately 10 cc are collected in a tube about  $\frac{1}{2}$  inch in diameter. The length of time required for the specimen to coagulate is the coagulation time. The norm by this method is eight to ten minutes.

The most accurate method is the one described by Howell. It is more complicated but the difficulties of technic are compensated for by its greater accuracy. The norm is eight to twelve minutes.

*The Bleeding Time*—The second test to be made in order to differentiate hemophilia from purpura is the bleeding time. The tip of the finger or lobe of the ear is pricked with a lancet and the drops of blood that appear are blotted away with a fresh clean blotter at one-half minute interval. The length of time required for the bleeding to stop is the bleeding time.

*Capillary Resistance* The third test is the capillary resistance test. A tourniquet is applied to the arm with a tension just short of obliterating the arterial pulse. After being kept in place for five minutes the constrictor is removed and the skin distal to the point of application is examined for petechiae. In severe cases the entire skin may assume a dusky hue due to myriads of petechiae whereas in milder cases the latter are few in number. It has been arbitrarily agreed that 10 petechiae comprise the minimum number for a positive capillary resistance test. When only a few are present they are most likely to appear on the flexor surface opposite the bend of the elbow. By applying the tourniquet so that it is just short of obliterating the pulse arterial pressure is transmitted through the capillaries to the veins. Normally the capillaries are able to withstand this pressure but in cases of purpura hemorrhagica rupture occurs and petechiae result therefrom. This does not happen in hemophilia.

*The Stick Reaction*—The fourth test is the stick reaction. If a needle is plunged into a vein of a patient with purpura hemorrhagica a small purpuric spot may appear in the layers of the skin at the site of the puncture. This does not result from extravasation of blood in the areolar tissue under the skin but is due to a true purpuric spot in the layers of the skin. The test is negative in hemophilia.

*Blood Platelets*—The final test is the blood platelet count. Whereas in hemophilia the normal count of approximately 300,000 obtains in purpura hemorrhagica the count is definitely diminished.

Although all hemophilia and purpura hemorrhagica cases do not fall sharply into the two groups indicated it is extremely important to differentiate the diseases both from a prognostic and therapeutic

standpoint Hemophilia is characterized by abnormal coagulation and the value of blood transfusion is not necessarily in direct proportion to the amount transfused. A sufficient amount is necessary but beyond that the value ceases. In purpura hæmorrhagica however, up to the point of complete control of the bleeding the value of the transfusion seems to be more or less in direct ratio to the amount transfused. Whereas in hemophilia one transfusion will frequently control the bleeding in purpura repeated transfusions are generally required. Supplementary roentgen-ray or radium treatment is also indicated in certain cases of purpura and at times splenectomy is advisable.

Although no case of purpura or hemophilia has ever been cured by blood transfusion, the bleeding symptom common to both can be controlled. Following such control, however, all have recurrent attacks. Between the latter, intramuscular injections of whole blood should be given once a month in amounts varying from 10 to 150 cc., depending upon the age of the patient. Although this type of therapy will not prevent recurrences it will reduce their frequency. Compatibility tests are unnecessary for the subcutaneous injections but one should be certain that the donor's Wassermann reaction is negative.

(d) *Leukemia*—Blood transfusion is of value only for certain symptoms of the disease. Although never curative in many instances it prolongs life through reducing or controlling bleeding and improving the patient's general condition. In fulminant types the patient succumbs rapidly whether transfused or not. Through erroneous deduction from such cases some observers state that transfusion is contraindicated in all acute cases of leukemia.

(e) *Bleeding in the New-born*—Blood transfusion not only controls bleeding in the new-born but in addition is a specific cure for the disease. When the hemorrhage occurs from the stomach or intestine, the indication for blood transfusion is more urgent than in any other type and the procedure should be performed promptly. A child who is almost exsanguinated can be immediately transformed into a rosey-cheeked healthy baby. When the bleeding is less severe, as in hemorrhage from the umbilical cord, circumcision or forceps injury, less radical measures may serve the purpose. Peroxide of hydrogen, alcohol tincture ferri chloride, Monsell's solution, and biologic agents such as 'Coagulose,' "Coagulin," 'Thromboplastin,' and "Thrombokinasé" will at times control the bleeding. Thromboplastin is probably the best of the group and should be applied for twenty minutes with pressure to the bleeding point after all clots have been removed.

Human, horse or rabbit serum, administered subcutaneously, may also be effective. Better than these, however is an intramuscular injection of whole human blood. If the bleeding continues after

such treatment it is wise to resort promptly to blood transfusion as within twenty-four hours a lethal loss may occur through oozing.

(f) **Rare Blood Diseases** This group includes such conditions as splenic anemia, hemolytic icterus, splenomegaly, etc. In these affections transfusion is of value chiefly as a supportive measure.

3. **Infections**—Blood transfusion is of great value in overcoming infections. Local pyogenic conditions as abscesses or osteomyelitis and wounds that are healing poorly are often greatly benefited by transfusion. The response is especially marked when secondary anemia is pronounced. Postoperative mastoid cases pursuing a septic course but with negative blood cultures can frequently be ended almost by crisis after one or more transfusions. In many quarters the tendency is to give small repeated transfusions for infections. While repeated transfusions are usually necessary there is no virtue in limiting the dose. It is better to follow the rule of giving as much as the patient can tolerate as often as required. This technic can be carried out best by employing whole unmodified blood.

Transfusion is frequently employed in *typhoid fever* and the blood is of greatest value if the donor has been immunized. It is especially indicated in cases of intestinal hemorrhage and preliminary to operation where there has been a perforation. Transfusion is of definite value in *erysipelas*. In *agranulocytosis* the procedure is a useful adjuvant particularly if there is an associated secondary anemia. It is extremely important that these patients maintain a high hemoglobin and red cell count.

In *pneumonia* blood transfusion is of especial value in children. With full appreciation of the fact that pneumonia is a self-limited disease and that its mortality average varies from year to year, our experience over a long period of time with a large group of cases is definitely convincing that transfusion in severe cases of bronchopneumonia is of inestimable value. This does not mean, however, that every case of bronchopneumonia should be transfused. In severe types blood is given on the second or third day of the disease whereas in less serious cases it is preferably withheld until the tenth to fourteenth day. In mild cases it is unnecessary if the progress is satisfactory. The erythrocytes and hemoglobin are estimated daily. The ideal case for transfusion is an extremely sick child with a progressively falling hemoglobin and the optimum time is the twelfth to fourteenth day of the disease.

For twenty-four to forty-eight hours prior to transfusion the pulse and respiratory rates are taken every two hours and then averaged. Following transfusion the same procedure is employed. If the post-transfusion pulse rate becomes slower it is a good prognostic sign and if in addition there is a slower respiratory rate

it is an excellent one. Conversely, an increased pulse rate is unfavorable and, in combination with an increased respiratory rate, ominous.

At times one blood transfusion will produce a crisis, in other cases several are necessary. Large doses of blood should always be given as small quantities are insufficient to replace the excessive amount of blood cells imprisoned in the consolidated pulmonic areas. If we consider the exudate in the lung to be blood, the child with pneumonia has virtually had a hemorrhage.

Bollinger has estimated that in the average case of bronchopneumonia an amount equal to 40 per cent of the total blood volume is contained in the pneumonic lung. Accordingly, these children should be given an amount of blood equal to 40 per cent of their total blood volume, estimating such volume as one-nineteenth of the normal body weight. The procedure does not in any way seem to control the incidence of complications, and the percentage of otitis media, mastoiditis, empyema, pyelitis, etc., is the same in transfused as in non-transfused children.

**Bacteriemia**—Transfusion is of greatest value in bacteriemia if the donor has been immunized with a vaccine prepared from the organism obtained from the patient's blood. The results from transfusion with blood of an ordinary donor have not been satisfactory. However, if the source of supply of the organism can be shut off, as by ligating the jugular vein in cases of sinus thrombosis, the results with ordinary blood are greatly improved. In cases where the bacteriemia persists after repeated transfusions, huge doses of vaccine should be administered to the donor in order to obtain adequate immunization. Daily injections should be given starting with 1,000,000,000 organisms and rapidly increasing the amount until a total of 150,000,000,000 to 200,000,000,000 is reached. After ten days of such treatment bacterial agglutinins usually can be demonstrated in the donor's serum in a titer of about 1 to 40 and the phagocytic index will be greatly increased.

The donor selected for immunization should be one whose blood is not only compatible with that of the patient but also whose phagocytic index is high. A donor who has been immunized by injecting a vaccine prepared from organisms isolated from the blood of the patient is termed an "autogenous immunized donor." If the vaccine is made from organisms obtained from the blood of another patient the donor is called a "heterogenous immunized donor."

When the diagnosis of bacteriemia is made the patient should be transfused with blood of a "heterogenous immunized donor" until the preparation of the "autogenous immunized donor" is completed. Transfusions from the latter donor should then be repeated until the blood cultures become negative. At times the temperature persists even after the bacteriemia has ended. This is generally due to the formation of abscesses and their appearance is a favor-

able prognostic sign. Following incision and drainage the temperature generally becomes normal.

4 **Toxemia** —Transfusion is of value in toxemias irrespective of the cause. It is of especial value if at the time of transfusion one performs a brisk phlebotomy or substitutive transfusion. The latter type is ideal in extensive burns and certain types of poisoning.

5 **Shock** —The value of transfusion in shock is in direct proportion to its timely administration following the onset of shock. Delay rapidly diminishes its effectiveness. Blood transfusion is also of definite value in preventing shock and the nearer the transfusion is performed to the time of operation the greater is its relative value. The usual practice is to transfuse the patient immediately before the anesthesia is started. Some believe the transfusion should precede the operation by three days in order to allow any untoward reaction which may follow to disappear. Although this may be indicated when citrate transfusions are performed it is unnecessary with whole unmodified blood as the incidence of reactions is extremely small. Furthermore a post transfusion chill never occurs while the patient is under the influence of ether.

6 **Miscellaneous Conditions** This group comprises certain conditions which have not been previously enumerated either because they occur rarely or are difficult of classification. The most important cases are those of difficult feeding and malnutrition. These frequently respond almost miraculously to one or more transfusions. Another condition which is helped by repeated transfusions is celiac disease. Lastly there is a group of cases which are transfused because everything else has been tried and found lacking. Actually they should not be transfused at all for it is just this type of case which tends to bring transfusion into disrepute. Patients who cannot possibly be helped by transfusion or anything else should not be given blood in the hope that it will work the impossible.

**Untoward Reactions** —The following untoward reactions may follow blood transfusion:

- 1 Fever
- 2 Chill
- 3 Urticaria
- 4 The result of an incompatible donor

1 **Fever** —This occurs in approximately 2 per cent of cases following transfusion by the Unger method in 10 to 20 per cent following other methods which employ whole unmodified blood and in 20 to 40 per cent of cases following citrate transfusions. Reactions should only be classified as febrile if there is a definite alteration in the character of the temperature following the transfusion and if the change raises the temperature  $1^{\circ}$  F. or more. The rise in temperature occurs in from one to two hours after the transfusion up to forty-eight hours thereafter. If the elevation occurs

at a later period, some cause other than the transfusion should be sought. Small doses of aspirin every two hours will overcome the temperature due to transfusion.

2 **Chill** — In children transfused by the Unger method chills occur in less than 2 per cent of the cases. The incidence appears higher following other techniques. The chill may occur during the transfusion or any time within forty-eight hours thereafter. It may vary from a slight chilly sensation to a severe and prolonged rigor. Morphine is useful in cutting short the reaction. A post-transfusion chill is always accompanied by a rise in temperature.

3 **Urticaria** — This untoward reaction may be very slight and the favorite site of the wheals in mild cases is around the eyes, lips and upper part of the chest. In severe reactions giant wheals may cover the entire body and become confluent. Severe coughing spells may result from urticaria of the trachea. Adrenalin given hypodermically promptly relieves the condition.

4 **Results Due to the Use of an Incompatible Donor** — After giving 20 to 100 cc. of blood, the patient if able to express himself, will immediately complain of pain in the small of the back. If the transfusion is promptly stopped the patient may possibly recover. If continued, the next complaint will be pain radiating down the legs followed by precordial distress, dyspnea and cyanosis, and in fatal cases, pulmonary edema. Morphine and alternating doses of adrenalin and atropine every fifteen minutes will at times save the patient's life. In such cases hemoglobinuria may follow and in some instances complete anuria. During convalescence small daily transfusions should be given.

**Relative Merits of Whole Unmodified Blood and Citrated Blood** — It is now recognized that the percentage of reactions following citrated blood is much higher than those following the transfusion of whole unmodified blood. There are also certain biologic differences in the two bloods. The addition of sodium citrate to blood diminishes its complement content. This is brought about in two ways: through a direct destructive action of the complement and by extracting from the red blood cell wall a substance which binds the complement. The first fact can be demonstrated by titrating the complement of the blood before and after citration and the second by performing a Wassermann test on citrated blood and noting that it has now become slightly anticomplementary. Furthermore, the addition of sodium citrate to blood diminishes the opsonins and reduces the phagocytic power of the white blood cells. For these reasons citrated blood is less valuable in cases of infection than whole unmodified blood.

Citrated blood is also less valuable than whole blood because it renders the red blood cells more fragile and more easily hemolyzed. In cases of blood dyscrasia in which more than one transfusion is

necessary whole blood is preferable for each time citrated blood is given there is a period during which the coagulation time is lengthened. Citrated blood is also undesirable in cases of nephritis. Complete anuria may occasionally result from overstimulation by the citrate. Lastly in extremely sick patients whole unmodified blood is definitely safer because sharp reactions seldom follow its administration. In general it may be stated that when normal blood is required to replace pathologic blood whole unmodified blood is indicated and when it is added merely to replenish an impoverished circulation citrated blood will serve the purpose.

**Selection of the Donor**—In order to select a donor whose blood is compatible with that of the patient the latter's blood must first be typed. There are various ways of doing this. Blood may be obtained from a vein or by skin puncture. It may be citrated or defibrinated and either serum or plasma may be used. In children it is better to prick the finger or lobe of the ear and thus keep the veins intact for the transfusion. Approximately 0.5 cc. of blood is collected in a small test tube containing a drop of 10 per cent sodium citrate in normal saline. A drop of this mixture is then diluted with normal saline solution. One drop of the diluted cells is put at the right hand end of a glass microscope slide and a second drop at the left hand end. To the left hand drop of cells a drop of known Group A serum is added and to the right hand drop of cells a drop of known Group B serum. Each specimen is then thoroughly mixed and covered with a cover slip. After five minutes the specimens are examined under the microscope for agglutination. If the picture is unchanged after fifteen minutes the result is considered final. Between observations the cells should be agitated by tapping the cover slip.

The patient may belong to one of four different groups. If the red blood cells of both specimens are clumped or agglutinated the patient is a Group AB. If the cells are not agglutinated by the A serum but are agglutinated by the B the patient is a Group A. If the cells are agglutinated by the A serum but not by the B the patient is a Group B. If the cells are not agglutinated by either sera the patient is a Group O. This method of naming the groups by letters is the International classification. The Jansky and Moss classifications employ numbers. The relationship between the three classifications is indicated by the following table.

Internat. onal. Jansky		Moss	Sera			
			AB	A	B	O
Cells	AB	4	1	0	+	+
	A	2	2	0	0	+
	B	3	3	0	+	0
	O	1	4	0	0	0

0 = no agglutination

++ = agglutination



Cross agglutination — After determining the group to which the patient's blood belongs similar tests are made on the blood of the donor. After finding one who is of the same group as the patient the cross agglutination or compatibility test should always be performed. For this test two more specimens are prepared. One is a mixture of the patient's serum and the donor's cells the other is a mixture of the donor's serum and the patient's cells. These should be examined microscopically and if both show no agglutination the donor is considered compatible and can with safety give blood to the patient.

It has been said repeatedly and even recently that this test can be omitted. The writer is definitely opposed to this opinion unless one is not adverse to having *unnecessary* reactions following transfusion. The control test should always be performed for the following reasons. It acts both as a check on the diagnosis of the patient's group and on the potency of the typing sera and also indicates whether or not the patient and donor belong to different subgroups.

Although all people belong to four main groups there are very occasionally individuals of the same group who show slight incompatibility. These can only be detected by performing the cross agglutination test. Although using such a donor will not cause the death of the patient a severe reaction with chill and fever may result. It is of equal interest to note that through error an absolutely incompatible donor—one belonging to a group other than that of the patient and not the so-called universal donor—has been employed without the occurrence of a reaction.

The Universal Donor — The Group O donor has been called the Universal Donor and the Group AB patient the Universal Recipient. These names have been applied because many believe that a Group O donor can give blood with safety to any patient regardless of his group and that the AB recipient can safely receive blood of a donor of any other group. The theory regarding their universality is in both instances the same. The donor's cells cannot be agglutinated because Group O cells cannot be agglutinated by the serum of any of the other three groups. Similarly the cells of the donor cannot be agglutinated by Group AB serum. With regard to the patient's cells freedom from agglutination is predicated on a different basis. Although under the microscope a Universal Donor's serum will agglutinate the cells of a patient belonging to another group and the Universal Recipient's cells will be agglutinated by the serum of a donor belonging to another group actually when the transfusion is given this does not occur. The agglutinins in the donor's blood are diluted by the patient's blood to such an extent that they cannot act. Furthermore they are limited in number by the amount of blood transfused and would

have to act on the huge number of cells contained in the patient's blood stream. These conditions are unfavorable for agglutination. The writer believes, however, that the circumstances unfavorable for agglutination do not always exist and in such cases agglutination will occur. If it does, it is just as dangerous as when the donor's cells are agglutinated. Such dire results can occur if the plasma of the Universal Donor (or Universal Recipient) contains agglutinins in a very high titer when the patient's erythrocytes are markedly diminished when the patient's blood volume has been greatly diminished through dehydration or severe hemorrhage or following massive transfusions.

If one is striving to surround the patient with every precaution and to give blood with the least possible disturbance and reaction, the donor should belong to the same group as the patient and every donor before every transfusion should be cross matched with the patient. This cannot be overstressed for even though a donor has been used successfully for one transfusion, he may prove incompatible for a second. In the interval between the transfusions the patient may develop minor agglutinins for the particular donor's cells.

**Compatibility of Mother's Blood** —It has been said that a mother's blood may be given safely to her new born without any preliminary test. This statement, however, has been found to be incorrect. Approximately 87 per cent of new born infants have neither agglutinins in their sera nor receptors in their cells. They therefore belong to none of the four established and recognized adult groups. Their group development may occur at any time up to four years of age, after which period the adult group becomes established and remains fixed for life. Should the patient in question be one of the 87 per cent, the mother's blood or for that matter any donor's may be used with impunity. The other 13 per cent, however, require testing and cross matching to exclude the danger of incompatibility. When typing the blood of a child belonging to the 87 per cent group, it will erroneously appear to belong to Group O. Therefore, whenever the blood cells of a new born infant appear to belong to Group O, the infant's serum should be tested against known Group A and Group B cells. If the serum agglutinates both A and B cells, the child is actually a Group O. If he belongs to the group of 87 per cent containing neither agglutinins nor receptors, the A and B cells will not be agglutinated.

**Rouleaux Formation** —There are two other conditions which may occur, namely, rouleaux formation and auto-agglutination. In the former, the red cells are piled on top of one another like a stack of coins. Under the low power of the microscope, an inexperienced observer may misconstrue rouleaux formation for agglutination. Under the high power, however, it should be unmistakable. Rouleaux almost always occurs in the donor's cells and but rarely in

the patient's. It is due to a rouleaux forming substance in the serum. The writer has transfused innumerable cases where the donor's cells have been rouleaued by the patient's serum without ill-effect. When the rouleaux forming substance is in the patient's serum it will act similarly on the blood of every donor tested and the risk of a reaction must be accepted or the transfusion omitted.

**Auto agglutination**—This is the agglutination of the patient's cells by the patient's serum. The clumping only takes place after the specimen of blood is taken from the patient and its temperature drops to less than body temperature. Under the microscope the cells appear exactly like agglutinated cells. They are not truly agglutinated however for if the temperature of the specimen is raised to  $37^{\circ}\text{C}$  the apparent agglutination disappears and the cells assume a normal appearance. This of course cannot be done with true agglutination. When auto-agglutination occurs the patient is erroneously typed as Group AB. Therefore when a diagnosis of Group AB is made it is wise to control this by examining a specimen of the patient's cells without the addition of any serum. If these appear agglutinated they must be washed several times with warm normal saline to remove the agglutinating substance. When the cells no longer show auto-agglutination they can be typed in the usual fashion and the correct group determined.

## METHODS OF BLOOD TRANSFUSION

The methods for performing blood transfusion can be divided into (a) direct methods and (b) indirect methods.

**Direct Methods**—Although no longer employed the direct methods are of historic interest. In the Carrel technic the artery was sutured directly to the vein. Theoretically this is an ideal procedure because it allows blood to flow directly from the artery into the vein and the blood touches nothing but the intima of the two vessels. The technic however is very difficult and in the hands of the average individual is almost always a failure. It was somewhat simplified by the introduction of Crile's cannula and this in turn was improved by Elsberg. Many other cannulae were devised and advised but the above were the most important and representative.

The second method was vein-to-vein anastomosis and various technics and cannulae were employed for its performance. All the direct methods have been discarded because they were difficult, uncertain and usually resulted in failure. It was impossible to know exactly how much blood was being transfused and in every case an incision of the skin of both the patient and the donor was necessary. This limited the number of transfusions the donor could give.

**Indirect Method**—These methods can also be divided into two groups (a) Those which supply whole unmodified blood and (b) those which supply modified blood

**Unmodified Blood** Although many methods have been devised only those of Kimpton and Brown Lindemann and Unger will be considered. These are chosen because they represent three distinct principles and are the original and best methods of their type

*The Kimpton and Brown method* utilizes a glass vessel which is a modification of the Percy tube. It is graduated and manufactured in various sizes. After the interior has been coated with a thin layer of solid paraffin the end of the tube is inserted into the donor's vein. As the blood wells up into the vessel the process is aided by producing negative pressure in the tube. When the desired amount has been obtained the tube is removed from the donor and by creating positive pressure at the top of the column of blood the latter is forced into the vein of the patient. It is a good method in that it supplies whole unmodified blood. The chief objection is that incisions of veins of both patient and donor are necessary and the blood may clot in the tube before it has been transfused.

*The Lindemann method* is a syringe cannula technique. A needle is inserted into the vein of the patient and the donor and by means of a dozen 20 cc. Record syringes blood is aspirated from the donor and immediately injected into the patient. Between the aspiration and injection of blood some saline should be forced through both needles. The method is good in that it supplies whole unmodified blood. Its chief objection is that the technique is fairly difficult and teamwork and dexterity are absolutely essential. Each time a syringe is connected to or disconnected from the needle there is danger of dislodging the needle from the vein. The production of a hematoma therefrom leads to technical difficulties.

*The Unger method* makes use of a special instrument. This consists of a stand to which is mounted a stopcock. The latter has two channels by means of which a Record syringe is alternately connected with donor and patient. When the syringe is connected with the donor for the aspiration of blood a syringe with saline solution is connected with the patient and vice versa. The continuous flushing with saline of the channel through which the blood is not passing insures freedom from clotting. The instrument is connected to the cannulae after they have been inserted through the skin into the veins of the patient and donor. A Record syringe is then inserted into the instrument and blood is aspirated. When the syringe is filled the stopcock is turned and the blood is injected. At the same time normal saline solution is forced through the channel which is not being used for the aspiration or the injection of blood. This procedure is carried on until the desired amount of blood is transfused. To prevent clotting in the syringe a stream of

ether is sprayed on its barrel while it is being filled or emptied of blood. In this way only one syringe is needed regardless of how much blood is being transfused.

Since the three methods were devised numerous others have appeared. Most of them are modifications and some brazen copies. The Unger method is the first which employed an instrument to transfuse whole unmodified blood. It has even been referred to as the direct method for blood transfusion. Although actually indirect it is the most direct of all the indirect methods. There are also instruments which utilize ball valves with or without springs copies of the Wechselsmann salvarsan apparatus. Such instruments are dangerous because the patient's blood can easily be given to the donor with the possibility of transmitting disease.

**Modified Blood** —The addition of sodium citrate to blood which is to be transfused has been used very extensively. Of the many methods suggested probably the best and the simplest is the one first described by Lewisohn. A needle is inserted into the vein of the donor and the blood is allowed to flow into a graduate into which 2 or 2.5 per cent sodium citrate solution has previously been placed. As the blood collects and mixes with the citrate it is stirred gently with a glass rod. The blood is collected in the proportion of 450 cc. to every 50 cc. of sodium citrate. This makes a final dilution of 0.2 or 0.25 per cent according to the citrate percentage used.

**Modified Stored Blood** —Whereas citrated blood has always been promptly given to the patient it has been advised recently to store the blood in an ice box for transfusion at a later date. Such blood can be kept for about two weeks or until it exhibits signs of hemolysis. After three days the leukocytes become reduced to about 300 and the erythrocytes undergo crenation. Only small amounts of such blood can be given with safety; larger doses yield a high percentage of reactions.

**Cadaver Blood** —Russian investigators have recently advocated the use of cadaver blood. Only individuals who have died suddenly from such conditions as trauma, coronary thrombosis or drowning are used as donors. From 2500 to 3500 cc. of blood are withdrawn from the external jugular vein within six hours of death. This blood promptly clots in the usual manner but within two hours it again becomes fluid and remains so. This remarkable and puzzling phenomenon of fibrinolysis occurs only in the cadaver blood of patients dying from sudden death; all other cadaver bloods clot and remain clotted.

Appropriate cadaver blood kept on ice remains suitable for transfusion for two weeks or occasionally longer. Large quantities may be administered without mishap; the percentage of reactions paralleling those of whole unmodified blood. The author has seen 4000 cc.

transfused into a patient within forty-eight hours. The chief drawbacks to the procedure are legal difficulties which interfere with obtaining the blood of a cadaver. An attractive feature is that it can be stored without the addition of sodium citrate.

**Site for the Injection of Blood**—The ideal site for giving blood is into a vein on the flexor surface of the elbow. While in adults an incision of the skin should never be made, in children it is necessary in about 5 per cent of cases. Before doing this, however, a thorough search should be made for an appropriate vein. It may be possible to enter a vein at the wrist, behind the internal malleolus, on the scalp or in the neck (the external jugular). The required incision should be  $\frac{1}{4}$  inch or less in size, made at right angles to the long axis of the arm and in one of the natural creases. By undermining the skin above and below the incision a piece of vein at least 1 cm. long can be retracted into view. A nick is then made in the side of the vein at a 45 degree angle with its long axis and the needle inserted therein. When the transfusion is completed, a dry dressing is applied without suture closure of the wound. All evidence of the incision soon disappears.

Transfusion of blood into the superior longitudinal sinus is a dangerous procedure in that the blood may be unknowingly injected into and around the brain. Some advise putting the blood into the peritoneal cavity. This is simply a makeshift and an admission of one's inability to perform the task intravenously. In addition to the danger of forming adhesions, there is the more serious hazard of perforating the intestine.

## CHAPTER VIII

### SURGICAL ASPECTS OF CERTAIN METABOLIC DISEASES

#### RICKETS

RICKETS (rachitis) is an infantile metabolic disease which results from a deficiency of vitamins C and D. It is characterized chiefly by the absorption of developed bone and the overdevelopment of calcium-deficient bone.

**Pathology.**—Specific bone changes predominate. The excessively vascular epiphyses become abnormally wide and thickened, the cartilage cells being increased in number and arranged in irregular columns. Due to deficient calcification and an excessive deposit of soft osteoid tissue, true bone formation is defective and irregular. Furthermore, there is marked absorption of developed bone and the lime content is decreased at times by 50 per cent. The bones thus softened are subject to deformities.

The periosteum also becomes hypervascular and the marrow exhibits an increase of erythrocytes and a diminution of myelocytes. The blood serum phosphorus is often considerably lowered and the condition may be associated with hypertrophy and hyperplasia of the parathyroid glands. With recovery, the bones become abnormally hard and brittle. The residual deformities are permanent.

**Symptomatology.**—The disease develops between the ages of three months and three years. Irritability and profuse sweating are early manifestations, sitting, standing, walking and dentition are delayed. The liver may enlarge and the abdomen protrude. There is also a tendency to colds and diarrheal attacks. Certain characteristic bone changes develop early: bending of the ribs from enlargement of the costochondral epiphyses (*rachitic rosary*), a transverse groove extending outward from the xiphoid cartilage, with the rib margins turned upward from diaphragmatic traction (*Harrison's groove*), and a vertical groove at the junction of the ribs and cartilage (*pigeon breast*).

The epiphyses of the long bones become enlarged, especially those of the lower ends of the radius, tibia and fibula. The softened weight bearing bones may bend, the commonest deformities being bowing of the lower third of the tibia (*bow-legs*), anterior curving of the femur, and at times *coxa vara* or *genu valgum*. The ligaments become relaxed, producing a tendency to kyphosis, flattening of the pelvis, dwarfing, and green-stick fracture.

The head is enlarged, lengthened anteroposteriorly, and flattened in the vertex; there is delay in closure of the fontanelles, bowing of the parietal and frontal eminences and craniotabes. The musculature also becomes weakened. Roentgen study of the distal end of the ulna commonly exhibits cupping and fraying (Fig. 4).



FIG. 4. Cupping and fraying of the distal end of the ulna.

**Treatment**—Breast feeding is the ideal treatment for young infants. Specific vitamin therapy comprises the administration of cod or halibut liver oils or their concentrates via terol and orange juice (vitamins A, B, C, and D). Natural or artificial lighthotherapy is also invaluable. Softened bones require rest and at times splinting or bracing becomes necessary. Following recovery, serious residual deformities may be corrected by osteoclasis, osteotomy or osteoplasty.

#### RENAL RICKETS

Renal rickets (renal epiphysitis, renal dwarfism) is a rare condition which occurs in children suffering from chronic interstitial nephritis or congenital polycystic kidneys. Growth arrest is usually evidenced before the age of five years. Painless displacement of the epiphyses of the knees, ankles, shoulders or wrists frequently



occurs in the eighth or ninth year and the progressive deformities resemble those of late rickets. Histologically the epiphysitis is less marked than in rickets the cartilage cells have a more orderly arrangement and the epiphyseal line is almost straight. Bone trabeculae are deficient in both osteoblastic cells and lime salts and general osteoporosis is demonstrable roentgenologically. Parathyroid hyperplasia commonly accompanies the condition.

The patients often exhibit infantilism and delayed sexual development. The urinary findings are those of chronic interstitial nephritis and renal insufficiency may be evidenced by dye and urea concentration tests. Nitrogen and phosphorus retention is usually present and the blood serum calcium is low. Death from renal incompetency generally occurs in the second decade. Corrective operative interference is contraindicated.

## SCURVY

Scurvy is a vitamin deficiency disorder. The condition may develop in children who are fed exclusively on certain proprietary foods or condensed sterilized or pasteurized milk. Susceptibility is probably also a factor. The importance of the disease from the surgeon's standpoint lies in its differentiation from certain surgical conditions.

**Symptomatology** — Approximately 80 per cent of the cases develop between the fifth and fifteenth months. The dominant symptoms are spongy bleeding gums, swelling and ecchymoses about the joints and extreme hyperesthesia. Tenderness of the legs is frequently the first symptom and the urine may contain red blood cells. Unless promptly checked by corrective diet the tenderness of the legs becomes acute and swellings develop about the joints. The mouth symptoms are seldom pronounced unless teeth are present. Bleeding may also occur from the nose, stomach or bowel. Pyrexia is present in severe cases.

**Roentgenologic Findings** — Spindle shaped thickening of the long bones due to subperiosteal hemorrhage is the dominant roentgenologic finding (Fig 5). The shadow may extend the entire length of the diaphysis and proximal to the epiphysis a transverse area of increased density may be present from which spurs protrude. If epiphyseal separation has occurred a zone of diminished density may be exhibited in the region of the metaphysis and the epiphyseal body may present a ring of increased density surrounding a rarefied central zone. Rickets is frequently associated with scurvy and the osseous pattern may be modified accordingly.

**Diagnosis** — This is based upon the history of vitamin deficiency, characteristic roentgenologic findings and prompt response to anti-

scorbutic feeding. The ecchymosis over the limbs associated with acute sensitiveness may suggest fracture or osteomyelitis. The lesions in scurvy are generally bilateral, are unaccompanied by hyperpyrexia or high leukocytosis and the roentgenograms are usually diagnostic.

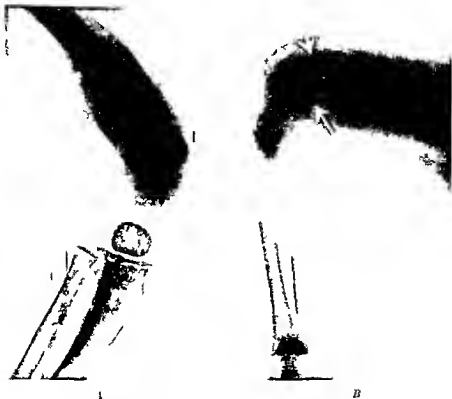


FIG. 5. A. Scurbutic subperiosteal hemorrhage. B. hemarthrosis.

Syphilitic epiphysitis may also simulate scurvy. The former is usually unilateral, rarely develops after the third month and is accompanied by other luetic manifestations including positive serologic findings. Sarcoma is occasionally suspected in late cases of subperiosteal hemorrhage when the lesion is unilateral. Careful examination, however, will usually disclose some evidence of bilateral involvement or of ecchymoses. In doubtful cases an antiscorbutic diet will clarify the diagnosis. A tumor may rarely develop from hemorrhage in the intestine and simulate intussusception.

## PART II

# CYSTS AND TUMORS

### CHAPTER IX

#### CYSTS

##### SEBACEOUS CYSTS (WENS)

SEBACEOUS cysts or wens seldom occur before puberty and are never congenital. They may develop anywhere in the skin where there are hair follicles and are most common on the scalp, face, neck, and back.

**Pathology** The cysts develop from dilatation of the alveoli of sebaceous glands and in rare instances from retention in sweat glands. The excretory duct is usually obstructed although at times sebum may be expressed. Its ostium frequently appears on the dome of the cyst as a black dot or dimple. Scalp wens, however, seldom exhibit either. The cyst wall is formed by the fibrous capsule of the distended gland and the lining membrane consists of flattened stratified or cuboidal epithelium which secretes sebaceous material. Some cysts have a laminated content, the result of epithelial exfoliation.

**Symptomatology** — Sebaceous cysts are often multiple. They are of slow growth, develop intradermally, elevate the skin in dome formation, and vary in size from a comedo to that of a baseball.

The tense, fluctuating tumefactions are covered by normal skin which is often adherent, especially at the site of the duct ostium. They occasionally become infected and the resulting abscess may eventuate in cure, sinus production, or recurrence of the wen.



FIG. 6. Unusual case of a sebaceous cyst in a boy of nine years.

**Diagnosis** —Sebaceous cysts seldom appear before puberty and are never found on the palms or soles. Their superficial intradermal character and common occurrence of a black dot at the occluded duct ostium readily differentiate them from dermoid cysts (fig 6). The latter are more deeply situated and occur at the sites of embryonic ectodermal fusion. (See Dermoid Cysts). Congenital epidermoids may be mistaken for sebaceous cysts although the latter are never present at birth.

**Treatment** —This comprises excision of the cyst wall with its entire lining membrane. Incomplete removal or simple puncture results in recurrence.

### DERMOID CYSTS

Dermoid cysts are endogenous teratomas and may be arranged in two genera: (1) Sequestration dermoids and (2) tubulo-dermoids.

**Sequestration Dermoids** —This type with the possible exception of the ovarian develops from a matrix of inclusion cells of the epiblast. The dislocation of these cells occurs either in situations where cutaneous surfaces coalesce or at the fusion sites of ectodermal with other structures. The cysts may be congenital or develop in early life. Others appear at puberty when the embryonal matrix is stimulated to growth by the extraordinary development of certain epiblastic structures which occurs at this period.

Although sequestration dermoids occasionally occur as skin lined recesses they are essentially globular cystic tumors lined with epidermis, dermis and dermal glands (skin). They commonly contain sebaceous material and hair, the character of the latter corresponding to that of the region affected. Teeth, cartilage bone and nerve fibers may be present in complicated cysts.

**Cephalic Dermoids** —Cephalic dermoids occur at the sites of coalescence of embryonic fissures: over the occipital protuberance, the anterior fontanelle, at the inner and outer angles of the orbit, in the tissues of the upper eyelid, over the mid line of the nose, at the junction of the nasal labial folds, just beyond the angles of the mouth and in the mid line of the floor of the mouth and mid line of the neck. These cephalic fusion zones are illustrated in Figs 7 and 8. In rare instances dermoids occur at the inner angle of the greater cornu of the hyoid bone. (The enlargement of a bursa which exists between the hyoid bone and the thyrohyoid membrane may be mistaken for a dermoid.)

**Dermoids of the Orbito-nasal Fissure** —These dermoids occur most frequently at the outer angle of the orbit. They form painless, rounded cystic tumors which are rarely larger than marbles and give the impression of being deeply attached. They are often connected with the periosteum and the underlying bone may be hollowed out. The site may be at the external angular process of

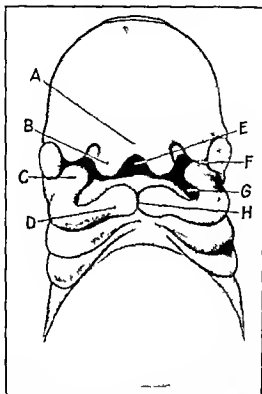


FIG 7 —Head of a human embryo about twenty days old *A* Fronto nasal *B* globular *C* maxillary and *D* mandibular process *E* internasal *F* orbito-nasal *G* mandibular and *H* intermandibular fissure

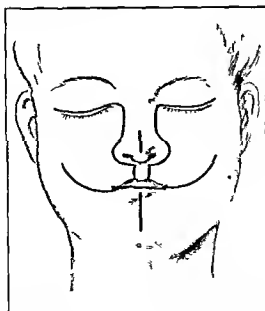


FIG 8 —Shaded lines indicate the sites of the embryonic fissures

the frontal bone or 1 to 2 cm posterior to it, or in rare instances beneath the eyebrow. Dermoids occur less often at the inner angle



FIG. 9 — Orbital dermoid

of the orbit and when deeply embedded may be attached to the dura. If pulsation occurs they may be mistaken for meningoceles.

(Fig. 9) Small dermoids developing in the upper eyelid arise in the fissure between the fronto-nasal plate and the skin fold which forms the lid; their lining at times possesses tactile sensibility.

Periorbital dermoids must be differentiated from sebaceous cysts, lipomas and subcutaneous cavernous angiomas. The first are superficial intradermal tumors and are uncommon before puberty. Lipomas are flattened and lobulated and are often connected with the skin. Cavernous angiomas are soft and velvety and their contents can be expressed by pressure.

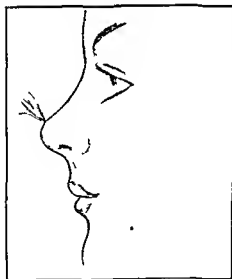


FIG. 10 — Dermoid in line of nose

**Nasal Dermoids** — Nasal dermoids derived from the orbito-nasal fissure and occurring in the nasolabial sulcus are rare. Teeth

have been reported in some. Those occurring near the tip of the nose the result of faulty fusion of the internal fissure may take the form of cysts although skin lined recesses furnished with hairs are more common (Fig 10)

**Auricular Dermoids** — Auricular dermoids develop at times from skin lined spaces left between the tubercles which unite to form the auricle they are commonly mistaken for sebaceous cysts. Others occupy the groove between the pinna and mastoid process and grow to the size of a cherry the underlying bone is frequently hollowed out

**Dermoids of the Trunk** — Dermoids of the trunk occur in the mid line of the body where the lateral halves of the fetus coalesce. This line of fusion may be described as beginning at the occipital protuberance and extending along the middle of the back to the coccyx passing through the perineum (scrotum and penis) and thence upward through the mid line of the abdomen thorax and neck to the margin of the lower lip

**Thoracic Dermoids** — Thoracic dermoids are rare and occur either within the chest or in the mid line over the sternum at the junction of the manubrium and gladiolus. A small skin lined sinus is more common (See Chapter XVIII)

**Dorsal Dermoids** — Dorsal dermoids are rare except in the sacro coccygeal region where they are often mistaken for spina bifida. Occasionally both conditions occur concomitantly. Dermoids also develop within the spinal canal in very rare instances

**Sacrococcygeal Dermoids** — In the caudal extremity complex embryonal processes form the basis of a series of fistulae cysts and tumors. The common postanal dimple is produced from vestigial remains of the neural canal. During the third month of fetal development the spinal cord reaches to the third coccygeal vertebra beyond which it is continued to the overlying skin as a fibrous cord containing groups of epithelial cells. With later development of the soft parts of the anus this cord atrophies and in so doing may produce a dimple termed the *fossa coccygea*. A superficial dimple has no surgical significance. When sufficiently deep however a *pilonidal sinus* lined with pilose epithelium may result (tubulo-dermoid). The secretion from sweat and sebaceous glands and the growth of hairs therein may be very annoying. This generally occurs at or soon after puberty. Another type of sacrococcygeal sinus or cyst may result from faulty coalescence of the lateral cutaneous layers (sequestration dermoid). In some instances the sinus tract may be only 1 or 2 cm in depth whereas in others it is deeply tortuous with several branches and may even pass ventrad to the coccyx and sacrum. A *pilonidal cyst* develops when the outer end of the sinus is closed.

In addition to the simple sacrococcygeal dermoids there is a

histologic potpourri of complex sacral dermoids, teratoid tumors, teratomas and fetal implantations. Cystic tumors lying on the ventral side of the coccyx containing intestinal wall elements with nerve tissue, may arise from vestigial remnants of the neurenteric canal. Others are attributed to remains of the postanal gut. They are present at birth and developing between the anus and coccyx, extend upward behind the rectum and displace the genitals. Teratoid tumors may be cystic or solid and occupy a similar situs.

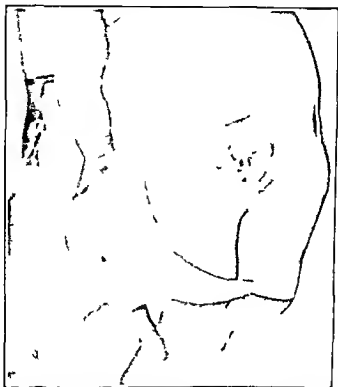


FIG. 11. Sacral teratoma. Also hydrocephalus, rectal prolapse and umbilical hernia.

**Sacral Teratomas**—Sacral teratomas in contradistinction to teratoid growths contain definite organs. They are usually bulky tumors lying on the dorsal surface of the sacrum and coccyx, and are often adherent to the periosteum. They may contain intestinal mucosa, gland structures, muscle, fat, cartilage, bone, nerve tissue, and rarely a rudimentary esophagus, stomach, intestine, pancreas or spleen. These tumors are fortunately rare and the infants are often still born. Mixed heterogeneous growths are potentially malignant. Bulky teratomas are frequently inoperable.

**Testicular Dermoids**—Testicular dermoids are pathologic curiosities. In most cases the tumors arise in the rete testis and contain greasy, sebaceous material and hair. They are generally present



at birth and may enlarge at puberty. Many growths reported as testicular dermoids have been sequestration cysts of the scrotum. Dermoids of the labium and penis are exceedingly rare.

**Treatment of Dermoids**—Simple dermoids may be excised at an optimum age of surgical competency but growing tumors require prompt removal. Small cysts in older children may be removed under novocaine (1 per cent for intradermal anesthesia and 0.5 per cent for the deeper parts), larger cysts and those adherent to deeper parts are best excised under general narcosis. (See chapter on Anesthesia.) Unless the cyst wall is completely removed, recurrence will follow. Aspiration and injections with escharotic solutions are futile.

Pilonidal cysts are generally easy of excision, pilonidal sinuses, however, require roentgenologic study following the injection of some radio-opaque substance such as lipiodol. Although the sinus appears superficial, it may be deep, circuitous and branching. Preliminary injection with methylene blue is an adjuvant in excising the tract. Frequent recurrences, due to incomplete removal, have popularized the injection of escharotics such as Cutler's solution. The results are sufficiently satisfactory to warrant a trial before considering surgery. If the epithelial cells are entirely destroyed by the caustic, permanent cure obtains.

**Ovarian Dermoids**—The pathology is considered in Chapter XXXVI.

**Tubulo-dermoids**—These are discussed in the chapter on Thyroglossal Cysts and Fistulae, and Branchial Cysts and Fistulae.

### EPIDERMOID CYSTS

Whereas the walls of simple dermoid cysts consist of epidermis, derma and dermal glands (skin), the lining membrane of epidermoids is composed solely of epidermis. This is usually well formed and often papillated. The cysts occasionally develop from sequestered embryonal epidermal cells but in most instances result from trauma.

**Congenital Epidermoids**—These growths are comparatively rare and occur as small single or multiple cysts of the skin (Fig. 12). Unless examined histologically, they are generally mistaken for sebaceous cysts. (It is probable that deep epitheliomas of the skin occurring in adult life arise from the epithelium of congenital epidermoids.)

**Implantation Epidermoids**—Implantation cysts develop from the invagination of epidermal cells following trauma and are usually found on the exposed surfaces of the fingers, palms and soles (Fig. 13).

**Diagnosis**—Congenital epidermoid cysts, commonly mistaken for sebaceous, never present a black dot at their domes. Their ultra-

mate diagnosis, however, often rests upon histologic examination. Implantation cysts reveal the evidence of a previous wound. Dermoid cysts are more deeply situated and occur at the sites of embryonic ectodermal fissures.



FIG. 12 — Congenital epidermoid cyst

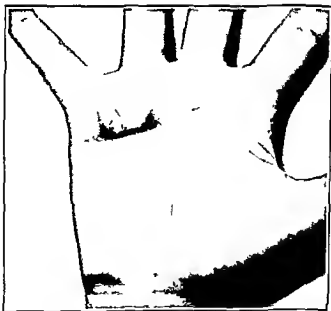


FIG. 13 — Implantation epidermoid cyst. Note scar

**Treatment** — This consists in excision of the cyst with its lining membrane.

## CHAPTER X

### TUMORS OF CHILDHOOD

TUMORS in children are relatively infrequent and the great majority are benign. Sarcoma comprises the usual malignant type carcinoma being encountered very rarely. The teratomas rhabdomyomas and tumors of the nervous system elements arising from remnants of undifferentiated tissue occupy a unique position. In some instances it is difficult to distinguish tumors from malformations.

Although no part of the body is immune certain regions are especially subject to the development of new growths. The kidneys and adrenals are most frequently involved and furnish approximately 30 per cent of the malignancies of childhood. The bones brain and meninges and the eye and orbit are the next most common sites and each accounts for approximately 10 per cent.

Most malignant tumors develop asymptotically. In many cases the appearance of an unusual swelling is the only symptom until the health becomes undermined through metastatic dissemination when rapid loss of weight and strength occur. The associated pyrexia at this period may suggest inflammation.

Benign tumors exhibit the same types and varieties as in adults. Those originating in the blood and lymph vessel are often congenital or develop soon after birth.

#### TUMORS OF THE BLOOD AND LYMPH VESSELS

Blood and lymph vessels are subject to neoplastic growth and produce tumors of two genera hemangioma and lymphangioma. The growths probably result from a developmental anomaly in the structure of certain vascular segments which retaining their embryonic character do not fit into the circulatory system and grow independently. This tissue predisposition occurs most often in the region of embryonal fissures and at times along the course of cutaneous nerves.

**Hemangioma**—These tumors also called angiomas are composed of abnormal formations of blood vessel. The clinical varieties comprise (1) simple nevus (2) cavernous angioma and (3) plexiform angioma or circoid aneurysm.

**Simple Nevus**—This variety occurs in several forms. *Nævus venosus* and *nævus flammeus* (port wine stain and strawberry patch)

are of common occurrence and consist of a circumscribed or diffuse dilatation and new growth of the superficial dermal capillaries and venules. The overlying epidermis being thin and transparent the dilated capillaries impart color to the skin. The red or bluish hue depends upon the preponderance of arterial or venous structure. *Telangiectasis* also a common variety consists of an abnormal collection of arterioles in the dermis or the subcutaneous tissue (Fig. 14). Spider nevus (*nevus araneus*) is composed of a central enlarged vessel with fine radiating capillaries which produce a web appearance.



FIG. 14. Congenital telangiectasis of the penna.

**Symptomatology.** Nevi are usually present at birth (birth marks) but at times appear during the first weeks of life or rarely later. Some are small and escape notice whereas others are very extensive and cover the side of the face or a large area of the trunk or an extremity (Fig. 15). They occur most commonly on the face, scalp, neck and back, less often on the limbs and rarely on the lips, tongue or conjunctivæ.

The clinical course is very variable. A nevus may completely disappear, remain the same size, grow slowly or rapidly, or take on vigorous growth after an indefinite quiescent period. It is essential to evaluate the relative growth of host and tumor and immediate treatment should be instituted when definite growth is evident. Simple nevus may develop into a cavernous angioma or large plexiform tumor and although essentially benign cause death from hemorrhage or septic thrombophlebitis.

**Diagnosis**—This is self evident in superficial forms. Unusual subcutaneous types may be mistaken for cysts. Disappearance of the mass upon pressure and its immediate refill upon release is characteristic of a vascular tumor. Aspiration will determine the diagnosis in puzzling cases.

**Treatment**—Many agents have been successfully employed such as carbon dioxide snow, ethyl chloride, caustics, radium and roentgen rays. Electric coagulation by either the Oudin or bipolar current has recently gained favor also. Obliteration by the injection of urea and quinine hydrochloride solution. (For an infant not over 0.12 gm. of quinine should be injected.) Radium produces the best cosmetic results. Radical treatment for extensive growths comprises excision with or without skin graft.

**Cavernous Angioma**—The growths are composed of connective tissue stroma supporting one or many endothelial lined sinuses comparable to the spongy erectile tissue of the penis. The sinuses intercommunicate and a main afferent and efferent vessel or vessels supply circulation to the growth.

**Symptomatology**—The tumors at times multiple are either present at birth or appear early in life. Occasionally they develop from simple nevi. Cutaneous tumors are most common occurring in the derma or subcutaneous tissues where the skin is loose, on the face, scalp, eyelids, buccal mucous membrane, lips, labium, scrotum and in folds about the buttocks and knees. They form distinct tumor fractions often rising above the surface and vary in color from red through crimson to blue. Deeply situated tumors are colorless and may be mistaken for cysts. The growths may remain stationary in size, grow slowly or vigorously or become circoid. Those occurring in the buccal



FIG. 15.—Unusual case of hematoma associated with pigmentosa. (Courtesy of Dr. T. N. Saxl.)

mucous membrane of the tongue and lower lip usually grow slowly but progressively and require early treatment. The angiomatous type of *macroglossia* is usually a combination of lymph and cavernous tissue (Fig 16).

In rare instances cavernous angiomas develop in the muscles liver spleen kidney uterus ovary or brain. They have even been noted in periosteum, bone-marrow and the myocardium. A rare group of metastasizing tumors also occurs which exhibit certain features of malignancy and eventually prove fatal from hemorrhage. Pulmonary metastases develop and other organs may become invaded. The primary growth and the metastases appear histologically benign.



FIG 16 Lymphangoma of tongue. Rapid enlargement after being present thirty years.

**Diagnosis**—Cavernous angiomas in the deeper structures may be puzzling. Obliteration upon compression with prompt refilling is helpful in diagnosis of aspiration.

**Treatment**—Small tumors may be successfully treated with carbon dioxide snow, radium, roentgen ray or electric coagulation. Larger growths require excision. Excessive bleeding may be minimized by preliminary ligation of the main supporting vessels.

**Plexiform Angioma or Cystoid Aneurysm**—In this rare type of tumor the abnormal vessels are arranged parallel to each other. The growth may consist of arteries or veins but usually comprises both. The vessels are often tortuous, especially the arterial and the mass may resemble a bunch of earth worms. Vessels of large caliber may pulsate and produce a bruit. The tumors occur

most commonly in the region of the temple scalp and perineum. Although occasionally congenital they generally develop during childhood from simpler types of angiomas. Those occurring on the scalp may partially erode the flat bones and communicate through newly formed foramina with the intracranial sinuses.

**Treatment**—If all simple angiomas were ablated early, circoid aneurysms would become pathologic curiosities as most cases develop from neglected small tumors. Surgical removal of these highly vascular growths is hazardous. Preliminary ligation of the main supporting vessels may be followed by subsequent endotherm excision in one or more stages.

**Melanoma**—This group of tumors is characterized by the formation of melanin. The benign form of moles termed pigmented nevus is often present at birth. They may occur as flat slightly elevated gray or almost black patches in the skin or be prominent roughened or irregular and marked by a growth of coarse hairs. Although most cases remain quiescent and become fibrosed through traumatism they may become malignant and spread by way of the blood or lymphatics to various parts of the body.

**Treatment**—Growths which are exposed to trauma or increase in size or become ulcerated should be promptly excised with a margin of healthy tissue by either the scalpel or endotherm knife.

**Lymphangioma**—These tumors are organoid structures consisting of endothelial cells and supporting connective tissue stroma both being involved in the neoplastic process and growing independently of the lymphatic system. Foci of round cells or lymph nodules occasionally accompany the growth. Akin to hemangioma the tumors contain lymph instead of blood.

Although many confusing names have been given to this genus of tumors such as hygroma, cystic hydroma, lymphoma, lymph adenoma and hydrocele of the neck, the clinical forms may be classified as follows: (1) *Lymphatic nevus*, (2) *cavernous lymphangioma* and (3) *lymphatic cysts*.

**Lymphatic Nevus or Lymphangioma Simplex**—This type occurs as colorless or slightly pinkish flat or wart like growths in the skin varying in size from mere specks to 2 cm. or more in diameter and consist of dilated lymphatics containing hyperplastic endothelium. They may be congenital or appear during childhood, occur most commonly on the face and neck and be single or multiple. They may also be found in the buccal mucous membrane of the lips and tongue appearing in the latter as pale pink papillæ which at times cover a large part of the lingual dorsum. Following trauma lymph may exude.

**Cavernous Lymphangioma**—The growths may develop in the skin, intermuscular septa or mucous membranes. They may be small and circumscribed or diffusely extensive and consist of dilated endothe-

lined lymph sinuses supported by a frail connective tissue stroma. At times they are composed of both vascular and lymph elements (hemolymphangioma). The tumors are usually congenital and occur



FIG 17—Macromelia

most commonly in the cheek, lip and tongue being designated respectively as *macromelia*, *macrocheilia* and *macroglossia*. The last rare condition manifests itself as a congenital enlargement of the tongue, the increase in size being due to lymphatic growth. The distal half is generally affected and the tongue may protrude. (Plexiform neuroma affecting the lingual and hypoglossal nerve may produce *microglossia*. An angiomatous type also occurs.)

**Treatment**—The treatment of simple and cavernous lymphangioma is the same as for hemangioma. (See Hemangioma.) It should be emphasized that cavernous lymphangiomas tend to slow but progressive growth and therefore require early treatment (Fig 16). Excision of large tumors is unsatisfactory, in many instances they are diffuse and invade deep and important structures (Fig 17). Moreover serious lymphorrhea may follow the division of large lymph radicals. Electric coagulation is a valuable therapeutic agent and portions of the tumor may be treated at different stages. Radium is seldom helpful. In *macroglossia* wedge excision of the distal part of the tongue has produced satisfactory results.

**Lymphatic Cysts**—These may be simple or multilocular. *Lymphangioma cysticum* are congenital multilocular cystic tumors which occur most often in the side of the neck and over the sacrum. They develop beneath the deep fascia and may be extensive at birth or grow to large size (Fig 18). *Hygroma colli* or lymph cysts occur in the anterior

most commonly in the cheek, lip and tongue being designated respectively as *macromelia*, *macrocheilia* and *macroglossia*. The last rare condition manifests itself as a congenital enlargement of the tongue, the increase in size being due to lymphatic growth. The distal half is generally affected and the tongue may protrude. (Plexiform neuroma affecting the lingual and hypoglossal nerve may produce *microglossia*. An angiomatous type also occurs.)

**Treatment**—The treatment of simple and cavernous lymphangioma is the same as for hemangioma. (See Hemangioma.) It should be emphasized that cavernous lymphangiomas tend to slow



FIG 18—Lymphangioma cysticum of the neck



triangles of the neck and may be congenital or develop during childhood or even in adult life. They are predominantly unilocular, may attain the size of an orange, and are translucent. Although situated beneath the deep fascia, they are freely movable and give the impression of being superficial (Fig 19). The tumor may extend upward as high as the parotid downward to the suprasternal notch, or rarely into the superior mediastinum or axilla. The cysts have a thin wall lined with endothelium and occasionally disappear spontaneously either with or without evidence of inflammation. Similar lymph cysts occur in the thoracic wall.

*Diagnosis* — Hygroma colli must be differentiated from branchiogenic, thyroglossal and dermoid cysts, cyst adenoma of the thyroid and suppurative lymphadenitis. Their location in the anterior triangles of the neck, superficial character, free mobility and translucency are salient characteristics.

*Treatment* — Lymph cysts occasionally disappear spontaneously before puberty. Palliative measures such as aspiration and injection of a few drops of tincture of iodine or 95 per cent carbolic acid, or irradiation by radium are at times curative. Unless the tumefaction is increasing in size, operation is best deferred until postpubescence. The entire cyst wall should be excised to prevent recurrence.



FIG 19 — Hygroma colli

*Retroperitoneal and Mesenteric Lymphangioma* — These occur as multilocular cystic tumors. Developing along the spinal column, they may grow downward behind the kidney and colon into the pelvis, upward toward the diaphragm, or into the mesentery and omentum. Preoperative diagnosis is speculative and complete excision is often impossible.

*Sacral Hygroma* — Sacral hygromas are rare cystic tumors which may develop in conjunction with spinal canal defects. Those containing nerve elements are probably aberrant meningoceles. Treatment comprises excision.

## FIBROMA

Fibromas are tumors composed of fibrous tissue. Their consistency varies from hard dense tissue in which the fibers are closely

interwoven to that of a wide-meshed alveolar structure filled with serum and resembling edematous tissue. Although fibrous tissue is often a conspicuous part of benign tumors pure fibromas are very uncommon.

The tumors develop at all ages and at times are congenital. Due to the generalized distribution of fibrous tissue they may occur in almost any situation. Hard types originate chiefly in subcutaneous connective tissue from ligamentous structures in the hands and feet and about joints and rarely from tendons or nerve sheaths. The soft variety occurs mostly in subcutaneous tissues and in the corium of the skin (fibroma molluscum). Both types may develop from the periosteum of the jaw (epulis) palate or base of the skull (nasopharyngeal polyp). Des-



FIG. 20 Pre-auricular fibroma

moids are peculiar fibrous tumors which grow in the muscular and tendinous parts of the abdominal wall. They may be diffuse, resemble sarcoma and tend to recur after removal.

**Symptomatology** — Fibromas of the hard type occur as painless circumscribed smooth or nodular masses. They are generally small, seldom attain a diameter of 2 cm and are usually freely movable (fig. 20). The soft variety develop jelly-like painless masses which are often pedunculated and covered with skin or mucous membrane. At times they undergo cystic degeneration and disappear.

Though essentially benign, fibromas occasionally recur after removal and in rare instances undergo sarcomatous degeneration. Metastatic fibromas have been reported.

**Fibroma Molluscum** These are curious skin tumors which occur commonly in adults and occasionally in children. The fibroma begins in the corium and projects through the epithelial layers forming a pedunculated growth. Due to inadequate blood supply the tumor may soften and disappear leaving a pedunculated skin tag. The latter at times desiccates and falls off.

**Fibromas of the Nerve Sheaths** — The tumors are rare and present two varieties. In one the fibroma develops in the nerve sheath and grows either upon the side of the nerve or involves the trunk. In the former instance the tumor may be enucleated without nerve damage whereas in the latter it is intimately associated with the

nerve fibers. The growths are usually of small size, seldom attain a diameter of 1 cm. and may be asymptomatic or exquisitely painful. Occasionally they are multiple. The second variety, termed *plexiform neuroma*, is of very rare occurrence and may be present at birth. The main trunk of the nerve is greatly enlarged due to fibromatous growth in the endoneurium. The nerve fibers are unchanged, however, and the perineurium remains intact. There is no pain or paralysis. At times the growth extends along the nerve to its filaments in the skin. The nerve trunk becomes lengthened and thickened and develops tortuosities resembling those of varicose veins. The tumors often recur after removal and may become sarcomatous.

**Treatment of Fibromas** — This comprises excision of the tumor. The ordinary fibroma is easily enucleated as it is circumscribed and often encapsulated. The capsule when present should be removed to prevent recurrence. Diffuse fibromas occurring in the metacarpal or metatarsal spaces demand meticulous excision as they are prone to recurrence and malignant degeneration. Fibroma molluscum are readily removed by scissors or the endotherm knife. Painful fibromas about nerve sheaths may necessitate the excision of a portion of the nerve trunk. Plexiform neuromas should be promptly and thoroughly removed as their growth is progressive.

## LIPOMA

Lipomas are tumors composed of fat tissue and their wide distribution is exceeded only by that of sarcoma. Arising most commonly in the subcutaneous and subserous tissues, they may develop in mucous and synovial membranes, in connection with periosteum, in and between muscles, and in the meninges of the brain and spinal cord. They are essentially tumors of adult life but their occurrence in childhood is not uncommon and they may be present at birth (Fig. 21).

Between the superficial and deep layers of the superficial fascia a fat layer is interposed. This adipose tissue is most abundant over the trunk and trunk ends of the body and it is in these sites that lipomas occur most frequently — the back, shoulders, neck, axilla and abdomen.

**Symptomatology** — Lipomas occur as firm, elastic, rounded, multiloculated growths without encapsulation but sharply circumscribed from the surrounding tissues. They vary in size from that of a pea to masses weighing several pounds and are often loosely adherent to the skin, causing the latter to pucker when the tumor is moved. Several tumors may occur in the same host. Although their consistency is generally that of normal fat, it may be reduced by secondary changes or increased through the admixture of fibrous

tissue (fibro-lipoma) or by metaplasia. The color of the growth is that of ordinary fat, yellow and translucent, with xanthomatous changes an orange tint may develop.

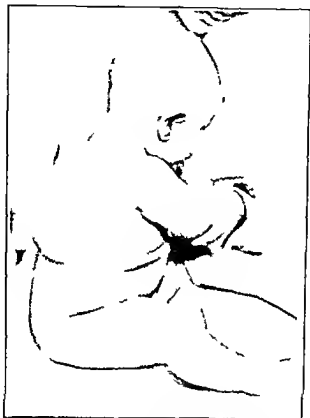


FIG. 21. Congenital lipoma of the back.

**Vascular Supply.** The blood supply of lipomas is abundant and each lobule grows about a separate branch of the nutrient vessel, thus producing an expansile but circumscribed growth. This unique type of blood supply may account for the size of lipomas remaining stationary when the body fats decrease in protracted illness; there is no peculiarity in their chemical composition which would prevent the fat mobilization factors from acting upon them. Occasionally an overgrowth of the blood vessels produces a vascular fatty tumor, *lipoma telangiecticum* or *carneousum*.

**Diagnosis.**—Superficial lipomas are readily diagnosed. Tumors containing abundant connective tissue (fibro-lipoma) or those in which salts have deposited are sometimes puzzling. Deeply seated growths may also cause perplexity. Small congenital lipomas

occupying the volar surface of the fingers and hands and connected with the tendon sheaths, may be mistaken for ganglia.

**Treatment.**—Lipomas are definitely benign tumors and may safely be ignored unless they enlarge or cause disfigurement or discomfort. In rare instances sarcomatous degeneration occurs in advanced life. Although the shelling out of a lipoma under local anesthesia is usually a simple procedure, the surgeon is often impressed with the abundant vascularity of the growth.

### MALIGNANT LYMPHOMATOSES.

The manifestations of this protean disease comprise Hodgkin's disease, pseudohygrota cysticum colli, lymphocytic leukemia, lymphocytic aleukemic leukemia, lymphosarcoma or pseudoleukemia, leukosarcoma, spindle-cell sarcoma of the lymph glands, endothelioma and mycosis fungoides. These diseases, primarily granulomatous, appear to be provoked by microorganisms in certain individuals whose lymph glands are specifically incompetent. Whether the inciting agent is a specific parasite, or a number of different bacteria, remains undetermined. Perhaps certain diphtheroid organisms may persist indefinitely in lymphomas as saprophytes and later become parasites through some inherent breakdown in the host's resistance. Atypical tubercle bacilli, especially the avian, are believed by L'Esperance to be a specific cause. Ewing also favors the tubercle bacillus or an allied organism. However, patients with malignant lymphomas seldom react to either old tuberculin or the avian variety.

**Hodgkin's Disease.**—Hodgkin's disease, no longer considered a clinical entity, is uncommon in infancy and childhood. Most cases occur between adolescence and early middle life. Although the specific etiology is unknown, it would appear that the parasites reach the glands from extralymphatic foci of chronic inflammation, either in the mucous membrane or skin. Such foci may occur about bad teeth, in the tonsils, sinuses, gastro-intestinal tract, lungs or skin. After an indefinite period of saprophytism, they acquire parasitic attributes and produce malignant adenopathies with attendant toxemia.

**Pathology.**—The glands may be enlarged to 3 cm. or more in diameter. Upon gross examination, the cut surface of a gland is opaque, yellowish-white and homogenous, the normal architecture being completely destroyed. Occasionally small necrotic foci are present. The capsule may be greatly thickened and periadenitis is common. At times the granuloma perforates the capsule and invades neighboring tissues. Histologically, the lesions are granulomatous and affect all the component gland elements. The lymphoblasts, at first overactive, become exhausted and atrophy

and the hyperplastic endothelium forms giant cells (Dorothy Reed). The stroma is increased and there is an eosinophilic infiltration which at times is pronounced. Rarely the granuloma degenerates into a sarcomatoid process. Practically any structure of the body may become involved except the central nervous system which is devoid of lymph elements.

**Hematology**—Slight erythrocytosis may occur at the onset in the late stage there is progressive erythropopenia and hemoglobinemia and in rare instances the blood picture may simulate pernicious anemia. The lymphocytes are at first increased and later decreased. Eosinophilia occurs in direct ratio to lymph tissue necrosis. In the subacute types immature lymphocytes predominate and occasionally they are found in such numbers as to suggest lymphocytic leukemia. The lymphocytosis is ephemeral however and is followed by lymphopenia. During the terminal stages of the disease a neutrophilic leukocytosis of 100 000 or more may occur. Sedimentation tests are increased in ratio to the acuity of the disease.

**Symptomatology**—The disease occurs in subacute and chronic forms and develops most often in the spring and fall when lymphatic competence is lowered. Some cases follow in the wake of the lymphotoxic diseases especially influenza. The clinical course of the disease is characterized by aggressive and regressive phases which ultimately become distinctly progressive with fatal termination. Depending upon the acuity of the process death may result after a few months or be delayed for many years. Recrudescences have followed apparent cures after a free interval of even twenty years. The average duration of the disease is three to five years.

**Mode of Onset**—The onset is usually in adenitis and cervical lymphomatosis is most common. The nodules develop asymptotically most often in the lower part of the posterior triangle of the neck. Although there may be an antecedent history of sore throats dental disturbances or influenza the previous history is usually negative. Primary involvement may also occur in the suprclavicular axillary inguinal mesenteric or meschiastinal glands.

If the primary adenopathy be of rapid growth there may be slight fever and malaise. Also during this aggression the glands may be rather soft confluent and sensitive to pressure. With development of the regressive phase the general health returns to normal and with subsidence of the peridematitis the glands become hard discrete non tender and freely movable. The acuity of the process varies considerably.

**Clinical Course in Subacute Types**—The lymphomas develop more rapidly and are prone to be multiple. Regressive phases are either brief or absent and there is intermittent remittent or continuous high fever. Case J I female aged thirteen year typifies the subacute type (Fig 22). Two months before admission the

child was in average health. She then developed anemia, malaise and lassitude. When presented for examination the patient was pale and undernourished, with temperature of  $102.4^{\circ}\text{F}$ . Physical examination revealed adenopathies in both posterior triangles of the neck and in the right axilla. The glands were the size of small marbles, soft, matted and quite tender. Biopsy of an axillary node confirmed the diagnosis of Hodgkin's disease. The process was actively progressive with continuous pyrexia of  $100.2^{\circ}$  to  $104.6^{\circ}\text{F}$ . Death resulted from cachexia in seven months. Fortunately such cases are rare.



FIG. 22.—Subacute type of Hodgkin's disease. Fatal in seven months.

*Clinical Course in Chronic Types*—The following history of P. D. illustrates the chronic type (Fig. 24). When fifteen years of age and apparently in excellent health the boy developed a left cervical lymphoma. The antecedent history was negative except for dental caries. When seen by the writer glands had been present for six months. Two months previously the patient had received roentgen-ray therapy with only slight improvement. Radical adenectomy was performed. The patient lived under an excellent hygienic dietetic regimen, and the subsequent regressive stage lasted six years. Glands then appeared in the right posterior triangle of the neck and right axilla. The nodes were tender, rather soft and matted. There was accompanying malaise and anorexia and the blood differential exhibited 4 per cent eosinophilia. Following two roentgen-ray exposures of high penetration, the glands subsided.

This further regressive stage lasted sixteen months. Lymphomas then developed in their former sites and in both inguinal regions.

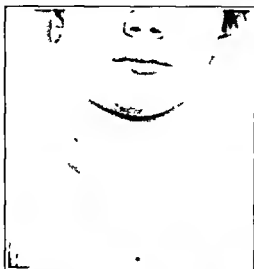


FIG. 23—Chronic type of Hodgkin's disease. Fatal in four years (Average duration)



FIG. 24—Chronic type of Hodgkin's disease. Fatal in eight years (Unusual duration)

The aggressive phase was precipitate and the tracheo-bronchial nodes became affected, causing racking cough and dyspnea. Six weeks before death the patient developed chylous ascites. The



blood in the terminal stages revealed a neutrophilic leukocytosis of 52,400. The total duration of the disease was eight years and four months.

*Aggressive and Regressive Phases*—The cycle of aggressive and regressive phases varies in each case. In the early regressive stages the patients may enjoy excellent health. During the aggressive phases, however, they are prone to feel under par and the glands become tender. Gradually the aggression becomes protracted, the constitutional symptoms more pronounced, and the patients begin to look sick. The lymphomas increase in size and others develop. The regressive phases become shorter and constitutional improvement is lacking. In the terminal stage, which may be explosive, lymphomas occur in multiple regions. The spleen may enlarge, chylous like fluid may occur in the pleural or peritoneal cavities, and occasionally an intractable pruritus develops. The terminal cachexia is febrile and often rapid.

*Treatment*—The prophylactic eradication of chronic foci of infection, such as diseased tonsils and teeth, probably salvages many potential cases. Possibly certain early cases of Hodgkin's disease are actually cured thereby. Once the disease is established, the treatment, like that of tuberculous adenitis, should be directed toward (1) increasing the patient's resistance, (2) removal of all extralymphatic foci of infection, and (3) local treatment of the adenopathy.

The general hygienic, dietetic treatment prescribed for tuberculosis of the cervical lymph nodes in Chapter XIX is equally important in Hodgkin's disease. In cases of severe anemia, repeated transfusions are supportive. Preoperative transfusion is often especially valuable. The serum of horses immunized against diphtheroid bacilli has proven efficacious in some cases; also the serum from another patient with Hodgkin's disease who has received radiation therapy. Arsenic and Colley's serum are valueless. Liver and its extracts are favored by Minot. The eradication of all foci of possible infection is imperative.

*Adenectomy*—Complete excision of the granuloma with extirpation of the neighboring nodes offers the best prospect for regional recovery and prolongation of life. Recutirpation of local recurrence is of no value at times. Radiation as a preoperative adjuvant may be advantageous in promoting inflammatory responses which reduce the peradenitis. Postoperatively it is contraindicated; it prevents healing and may be actually harmful. Inoperable and recurrent cases are best treated by roentgen ray irradiation. A few large doses of high penetration administered at infrequent intervals produce the best results. By the judicious combination of surgery, radiation, and a careful health regimen, patients may have several

years added to their prognosticated expectancy. (Some clinicians oppose adenectomy.)

*Pseudohygrota Cysticum Colli*—This very rare condition manifests itself in early infancy by the development of nodules and diffuse areas of fluctuation in the lower part of the neck. Multiple transfusions may prolong life. Surgery, however, is contra indicated.

**Lymphocytic Leukemia, Lymphocytic Aleukemic Leukemia, Lymphosarcoma and Leukosarcoma**—Although each has been



FIG. 3. Lymphosarcoma of the cervical and mesenteric nodes.

described as a separate entity, they appear to be responses to similar stimuli and like Hodgkin's disease pursue a subacute or chronic course.

In the subacute types of lymphocytic and aleukemic leukemia a fulminating purpura may be the first symptom. Lymphomas develop in the superficial glands, liver and spleen. They tend to be large, soft, tender and confluent. In the lymphocytic type there is hyperlymphocytosis whereas in the aleukemic the lymphocytosis is only moderate. In the chronic forms lymphomas develop slowly.

and remain discrete and non tender. The lymphocytes in both types are composed mainly of small mature cells thereby differing from Hodgkin's disease. The metabolic basal rate may be greatly augmented. Treatment comprises supportive measures and irradiation. The latter usually reduces the metabolic rate. Both diseases progress to fatal termination.

**Lymphosarcoma**—The disease is rare in early life and generally occurs in the lymph tissue of the gastro intestinal tract (fig 25). The lymphomas grow rapidly, often produce intense periadenitis and invade surrounding structures. The process however is distinctly granulomatous and not neoplastic. The diagnosis is seldom made preoperatively. Excision of the mass with the neighboring mesenteric and retroperitoneal glands, sometimes requiring intestinal resection, offers the best prospect of prolonging life. In inoperable cases radiation may retard the activity of the process.

**Leukosarcoma**—Leukosarcoma is a very rare condition about which little is known. It is characterized by persistent hyperlymphocytosis and regional lymphomatosis.

**Spindle cell Sarcoma of the Lymph Glands**—This is the rarest form of chronic Hodgkin's disease. The lymphomas are composed of dense fibrous tissue including therein a few lymphocytes, endothelial cells and an occasional eosinophile. The clinical course is one of chronicity and except for the slowly progressive adenopathy, the patients appear and feel well until the terminal phase. Extirpation of the glands except in very early cases is useless. Radiation is ineffective.

**Endothelioma of the Lymph Nodes**—This rare neoplastic disease is herewith noted because of its similarity to Hodgkin's granuloma. Clinically the disease occurs either as a systemic involvement of many lymph nodes with progressive fatal termination or as a single or multiple locally aggressive recurrent malignant tumor. Treatment comprises radical excision or radiation.

**Mycosis Fungoides**—Mycosis fungoides is a form of lymphocytic leukemia in which lymphomas of the skin are the dominant factor. The tumors are usually small and discrete and the overlying skin deep red or purplish. The disease is rare especially in childhood. Radiation is recommended.

## PART III.

# DISEASES OF THE OSSEOUS SYSTEM.

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## CHAPTER XI.

### DEVELOPMENTAL DISEASES OF THE BONES

**Bone Development.**—The bones of the trunk, limbs and base of the skull are preformed in hyaline cartilage and remarkable tissue changes occur in their metamorphosis. An ossification area appears in the center of the cartilage of the long bones about the fortieth day of fetal development and gradually extends in both directions of the shaft. At birth, or within the first six months of life, complementary centers of ossification begin to appear in each end of the cartilage. As the child develops, ossification progresses until there remain of the original hyaline cartilage only two thin discs separating the bony shaft, or diaphysis, from the ossified ends, or epiphyses. The discs are termed the epiphyseal plates or conjugal cartilages and the portion of the shaft adjacent to them, the metaphysis. Final ossification of the epiphyseal cartilages occurs at different periods in various bones and the entire process is not completed until the twenty-first to twenty-third year. (See table under Sporadic Cretinism.)

**Bone Growth.**—Growth, accretion and ossification are greatly influenced, if not entirely controlled, by the internal secretions of the thyroid, parathyroid and pituitary glands. Growth in the longitudinal axis occurs from the epiphyseal plates but the growth rate is not the same in both ends. It is more rapid in the upper epiphysis of the humerus and lower epiphyses of the radius and ulna, and in the lower epiphysis of the femur and upper epiphyses of the tibia and fibula. Growth in the transverse diameter results from a combination of accretion and absorption. Concomitantly with the laying down of new lamellæ between the periosteum and cortex, the medullary cavity is enlarged through the absorption of cancellous tissue adjacent to the endosteum. The accretion is produced chiefly by the periosteal osteoblasts, and absorption by the osteoclasts which lie between the endosteum and bordering cancellous tissue. (For further details refer to Anatomy of Bone in Chapter XII.)

## ACHONDROPLASIA

(CHONDRODYSSTROPHIA FETALIS)

Achondroplasia is a congenital abnormality of bone formation from cartilage. The deficiency of endochondral ossification in the epiphyses produces a marked diminution in growth, form and size of the long bones. The subjects have long bodies, large heads and short limbs.

**Etiology**—The cause or causes remain undetermined. Some investigators attribute it to fetal rickets. The condition however, differs from rickets in being aplastic rather than hyperplastic also in the excessive ingrowth of connective tissue. Others favor thyroid deficiency and congenital lues as causative factors. Neither appears tenable.



**Pathology**—The condition is the end product of a deficiency of endochondral ossification in the epiphyses. The head is large, with small face and pug nose due to normal development of the membranous bones and inhibited growth of the procartilaginous. The trunk is of normal size but the extremity bones especially the humerus and femur are very short producing a diminutive stature of 3 to 4 feet. The hands are stubby and the fingers diverge (*trident hand*), the sacrum is tilted forward almost to a horizontal position causing an apparent lordosis and protuberant abdomen. The head of the femur is small with attendant coxa

tive tissue may also occur at the junction of the metaphysis with the diaphysis. Periosteal bone formation remains normal.

**Symptomatology.**—The typical achondroplastic dwarf (the King's jester) is readily distinguishable—short stature, large head, small face, pug nose, short extremities, especially the arms and thighs, long trunk, lordosis, protuberant abdomen, bowed legs, and stubby hands with trident fingers. The musculature is well developed. Although they appear stupid, mental competency is unimpaired. Roentgenologically, the short stubby bones exhibit broad epiphyses and the epiphyseal cartilage plates are uneven and flare outward. The vertebrae are wedge-shaped and the intravertebral spaces widened. The *coxa vara* and small femoral head aid in diagnosis.

**Treatment.**—There is no specific therapy. Vitamins and thyroid extract have been recommended. Deformities may occasionally require orthopedic correction.

## SPORADIC CRETINISM

### (MYXEDEMA OR HYPOTHYROIDISM)

Sporadic cretinism may be congenital or develop at any period of life. The congenital type occurs in infants born without a thyroid gland, whereas the infantile form, which develops most commonly during the fifth and sixth years, results from postnatal thyroid insufficiency. The condition occurs infrequently and the etiology is undetermined.

**Pathology.**—The thyroid gland is always the site of degenerative changes of variable degree. In congenital cases the gland may be totally absent or very rudimentary. In infantile types it is usually small and exhibits atrophic acini, diminished colloid and fibrous tissue infiltration. At times the hypophysis is enlarged.

**Symptomatology.**—The disease is characterized by a diminution of all vital processes and by certain trophic disturbances. There is a slowing down of metabolism and a diminished excitability of the vegetative nervous system. Mentally and physically the organism remains at the stage of development attained before the onset of the thyroid degeneration. Thyroxin is supplied in mother's milk, and upon weaning an infant with thyroplasia, physical and mental growth cessate promptly. In infantile cretinism, normal development proceeds to the time of onset of thyroid incompetency.

**Thyroxin.**—The function of thyroxin, the active principle of the thyroid, appears to be that of a catalyzer. By its presence, the amino-acids which result from protein metabolism are reduced to carbon dioxide, water and ammonium carbonate, the latter being converted into urea by parathyroidal action. Deficiency of the activating thyroxin results in the retention of nitrogenous waste

products. Lack of metabolic activity affects all the cellular elements of the organism and concomitant sluggishness occurs in the secretory production of other endocrines. Thus through polyglandular insufficiency hypopituitary and hypoadrenal states may be engrafted upon the hypothyroidal.

**Effect Upon Bone Development**—Thyroid deficiency in early life produces cretinoid states with secondary eumelioid characteristics. One of the chief effects is cessation or marked diminution of bone formation and bone growth in all parts of the osseous system which originate in cartilage.<sup>1</sup> A delicate small skeleton results in which ossification of the epiphyses is markedly delayed.

The following table indicates the ages at which ossification centers normally appear in the wrist and hand.

<i>Age</i>	<i>Appearance of Bone Shadows in Roentgenograms</i>
At birth	Distal halves of phalanges metacarpals and ulna
4 to 8 mos	Ossification in aniline form
1 to 2 yrs	Lower epiphyses of radius
" 3	Epiphyses of proximal phalanges
2 1/2 to 3	Epiphyses of other phalanges
4 to 5	Semilunar
5 to "	Trapezium trapezoid scaphoid and lower ulnar epiphyses
10	Pisiform
13	Sesamoid bones
16 to 17	Disappearance of epiphyseal lines of phalanges and metacarp
20 to "	Disappearance of all epiphyseal lines

The roentgenogram of the hand of a cretin of seven years (Fig. 27) typifies the generalized osseous delay. The shadows of ossification are comparable to those which normally appear in a child of two years. The membranous bones, however, escape the dystrophy. Thus the head may develop to normal size and supported upon a frail diminutive body give the impression of hydrocephalus.

**Myxedema**—This condition is not a dominant characteristic of cretinism. A portion or all of the skin may be affected especially that of the face neck hands and feet. The thickened indurated skin is dry rough and at times heavily folded. Fat pads develop in the supraclavicular fossa and over the hips and abdomen. The backs of the hands and feet may be puffy and the former appear spade-like. The hair is coarse and brittle and falls out readily. The fingernails grow slowly and are often cracked and scamed. Sweat and mucous secretion are markedly diminished. The tongue is often large and protruding and the teeth are late in erupting. The fontanelles remain open and walking is delayed. In

<sup>1</sup>The administration of thyroid extract in cases of delayed ossification as a corrective is indicated upon this hypothesis.

pronounced cases there may be extreme constipation due to bowel atony

**Metabolism** —Body metabolism is at its lowest point with attendant slow pulse and subnormal temperature. The basal metabolic rate may even exceed  $-60$ . There is lack of mental development and a stupidity which at times degenerates into a state akin to hibernation. Even under appropriate treatment many cretins never attain the mentality of puberty and their sex organs remain infantile or fail to fully develop.

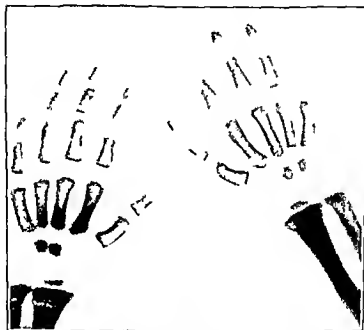


Fig. 27 Delayed ossification in cretin of seven years

**Treatment** —The miraculous effects of early treatment with thyroid extract in congenital thyroplasia is the outstanding triumph of glandular therapy. In older children pituitary extract and iodine are at times valuable adjuncts. Many cases are unfortunately overlooked for long periods and irreparable developmental damage results.

### ENDEMIC CRETINISM

Endemic cretinism is a chronic endemic disease characterized by disturbances of growth, enlargement of the thyroid gland, deficient mental competency, and at times deaf mutism. There are undoubtedly other factors than thyroid insufficiency in its genesis and the specific etiology is undetermined. The cases occur chiefly in the



intensely gortrous districts of the Alps Pyrenees and Carpathians and there is usually a striking familial history of cretine degeneration.

**Symptomatology** — The chief characteristic is dwarfism associated with a large head of quadrangular shape. The face is prognathic, the bridge of the nose flat, the eyes widely separated and the lips large and thickened. The skin of the face is loose and wrinkled and the individuals appear sad and prematurely old. The dwarf stature results from interference of bone growth and retardation of ossification. The extremity bones and ribs are short and thickened and scoliosis and ankylosis are common.

The skin may or may not be myxedematous. The intelligence is always subnormal with wide variations from slight incompetency to idiocy. Sex development is retarded and deaf mutism is not infrequent. The basal metabolic rate is subnormal. Although the thyroid is generally enlarged, in some cases it appears normal and the role if any which the gland plays in the genesis of endemic cretinism is undetermined.

**Treatment** — Where as some cases of endemic cretinism are improved but not cured by the continuous administration of thyroid extract the majority are uninfluenced. The condition concerns the internist and orthopedist rather than the surgeon.

epithelium (tubulo-dermoids) or from ectodermal rests sequestered with closure of the cerebral vesicles (sequestration dermoids). Complex teratomas have also been described. They probably develop by metaplasia from hypophyseal duct remnants. Solid tumors of the hypophysis benign and malignant occur only in adult life.

**Endocrine Function**—Since Marie's description of acromegaly in 1885 much has been learned concerning the internal secretory function of the hypophysis. It would appear that the gland is necessary to life throughout the vertebrate kingdom and that its internal secretion exercises a dominant influence on body development and growth.

Hypofunction of the anterior lobe leads to an abnormal deposition of fat, persistence of the infantile type of genitalia, amenorrhea at pubescence and occasionally hyperglycemia and glycosuria. In adults there may be loss of sexual power in the male (pituitary eunuchism) and amenorrhea in the female.

**Gigantism and Acromegaly**—Hyperfunction of the anterior lobe during prepubertal and adolescent years resultant upon hypertrophy or adenoma may produce gigantism. When however the hyperfunction develops in adult life after closure of the epiphyses inhibits further longitudinal growth the hand and soft tissues of the head, face, hands and feet may become affected and result in acromegaly. Thus gigantism and acromegaly are both produced by oversecretion of the anterior lobe, the result depending upon whether the dysfunction occurs before or after obliteration of the epiphyseal cartilages. (Recent studies would indicate that acromegalic changes may even begin in the second decade.)

The combination of gigantism and acromegaly is an evidence of long continued hormonal overproduction beginning in childhood and continuing unchecked into maturity. Ultimately the gland tends to undergo functional involution to a state of hormonal failure.

Although precocious physical development, sexual and somatic may result solely from pituitary dysfunction, it occurs more often with adrenal or thymic pathology or without discoverable lesions of the endocrines.

**Treatment**—The therapy of anterior pituitary disease in children is still experimental. Many cases are end products of polyglandular dysfunction.

## OSTEOMALACIA

### (MOLLITIES OSSIUM)

Osteomalacia is a softening of the bones due either to progressive decalcification or to inadequate deposition of lime salts. In early life the condition occurs chiefly as a secondary manifestation in rickets, scurvy and osteitis fibrosa.

The disease occurs endemically in Switzerland and the Rhine provinces and exhibits seasonal exacerbations; it is also prevalent in China. Over 90 per cent of the cases occur in females. The pathogenesis is undetermined. Hypothetical factors include diet, pregnancy, lactation, adrenal insufficiency and parathyroid dysfunction.

**Pathology**—Decalcification occurs chiefly in the bones of the pelvis, spine, thorax and extremities. As softening progresses, marked deformities may develop in the weight-bearing bones. The cortex thins and the marrow widens and fracture may occur in cases of extensive decalcification. Histologically, the Haversian canals are dilated, the bone cells enlarged, irregular and crowded and the lamellæ obscured. The new osteoid tissue exhibits deficient ossification and thinning of the trabeculae. The blood serum calcium is often lowered and excessive calcium is excreted in the urine. There may also be an associated enlargement of the parathyroids.

**Symptomatology**—Progressive bowing of the long weight-bearing bones, spinal curvature and the development of a waddling gait are the chief symptoms. There may also be mild pain and weakness in the affected bones. Spontaneous fracture is common. Roentgenologically, the bones exhibit rarefaction with thinning of the cortex and widening of the medulla. Extensive lesions may resemble the diffuse type of osteitis fibrosa cystica (Von Recklinghausen's disease).

**Treatment**—Cases associated with rickets or scurvy improve with appropriate treatment of the primary disease. Heliotherapy, high-calcium diet, cod or halibut liver oils or their concentrates, vitamin D and the oral administration of calcium and phosphorus are recommended adjuvants. Orthopedic appliances may be required for support or correction. Glandular therapy is experimental.

## FRAGILITAS OSSIUM

### (BRITTLE BONE)

Many diseases, such as rickets, infantile scurvy, osteitis fibrosa, hyperparathyroidism and other conditions characterized by bone brittleness and fracture tendency, have been discussed under a potpourri of old terms. For clarity and simplicity, the varied pathologic conditions are presented under (1) Osteogenesis imperfecta and (2) Osteoklasi.

**Osteogenesis Imperfecta** (Osteoparhrosis, Fragili Ossium) is a pathologic condition in which the bones become abnormally brittle with a tendency to multiple fracture.

**Etiology**—Although the cause is unknown, it would appear to be some dysfunction of the endocrine secretions which control the physiology of bone growth and ossification.

**Symptomatology** — The condition may be present at birth develop in infancy or be delayed until puberty or early adult life. The congenital type is most common and the changes which occur during intra uterine life often result in stillbirth. The defective formation of osseous tissue affects both membranous and cartilaginous bones. The extremity bones are long and thin extremely brittle and break under slight stress. The cortex is thin the lamellæ being irregularly formed and partly replaced by cancellous structure. The marrow is either fatty or fibrous. The skull may exhibit frontal and supra-orbital prominences an underhung jaw and a tilting downward of the axes of the orbital and auditory canals. The pipe stem character of the bones is readily demonstrable roentgenologically.

In the infantile type nothing abnormal is noticed until the child begins to walk and tumble and fractures occur from trivial injuries. Union occurs rapidly through excessive callous formation and growth rate is unimpaired unless the epiphyseal plates are damaged. The tendency to fracture diminishes with age and generally disappears after childhood (Fig 28).

The pubescent type exhibits similar symptoms and is often hereditary. Most cases have blue scleræ. The stature may be short and the joints hypermobile. Deafness may occur in early adult life. Roentgen study exhibits either eccentric atrophy with a thinned cortex and widened medullary cavity or concentric atrophy with a thinned cortex and narrowed marrow cavity.

**Diagnosis** — The roentgenologic findings of a thin rarefied cortex with fractures in various stages of healing is pathognomonic. Stillbirths may be confused with syphilis. In the latter the skin is macerated there is acute epiphysitis and the placenta exhibits definite changes. Rickets is readily differentiated from the infantile type by the saucer shaped epiphyseal plates rachitic rosary and



Fig 28 — Osteogenesis imperfecta

Harrison's grooves. The china blue sclera is a striking characteristic in most cases of osteogenesis imperfecta.

**Treatment**—There is no specific therapy. Phemister advocates the use of phosphorus. Others recommend cod- or halibut liver oil or their concentrates, viosterol, calcium and thyroid extract.

**Osteosclerosis (Marble Bone, Spotted Bone, Albers-Schönberg Disease)**—Osteosclerosis is a rare nonhereditary condition in which there is both an increased density and fragility of the bones. The histogenesis remains obscure. It has been attributed to a primary rachitic-osteomalacic basis and also to endocrine dysfunction.

**Pathology** This varies with the stage of the disease. Early cases exhibit an irregular condensing osteitis, certain bone areas being eburnated while others remain normal (spotted bone). In advanced cases the bones present a uniform dense homogenous structure with obliteration of bone architecture (marble bone). Histologically, the periosteal osteogenic tissue is largely replaced by fibrous tissue, the cortex contains but few bone corpuscles and the medulla is replaced by dense bone. Although the epiphyses are similarly involved and the epiphyseal cartilage plates contain irregular areas of calcified bone, the formation of new bone is unaffected and there is no dwarfing.

**Symptomatology**—The disease may occur at any age but is most common at prepubescence. Congenital cases have been reported. It produces no symptoms and fracture following casual injury generally attracts attention to the pathology. Despite the paucity of osteoblastic elements osseous repair proceeds in normal manner. In rare instances optic atrophy develops from narrowing of the foramina. The blood chemistry is unaltered. Leukemic changes occur in some cases.

**Treatment**—There is no specific treatment. Cod- and halibut-liver oils or their concentrates, viosterol and dietary regimens have been ineffective, likewise parathormone.

### CALCINOSIS UNIVERSALIS

**Calcinosis universalis** is a rare disease which occurs chiefly in the first two decades of life. It is characterized by (1) extensive deposits of calcium in the subcutaneous tissues, (2) scleroderma and (3) peripheral vasospasm resembling Raynaud's disease.

**Etiology**—Although the pathogenesis remains undetermined the disease appears to be related to dysfunction of the parathyroid glands. The latter definitely influence calcium metabolism and effect a maintenance of normal calcium in the osseous system and in the blood serum. Hyperparathyroidism is associated with the extraction of calcium from the skeleton, an increase of calcium concentration in the blood and urine, and a diminution of serum phos-

phorus (Possibly the thyroid also influences the physiology of calcium metabolism)

Although in *calcinosis universalis* no alteration occurs in the serum calcium similar normal values for calcium have been reported in several cases of clinical hyperparathyroidism. Generalized osteoporosis develops concomitantly with calcium deposition in the subcutaneous tissues. The added association of scleroderma and vasospasm further suggests parathyroid dysfunction as an etiologic factor.

**Symptomatology**—Calcium salts are deposited in the subcutaneous tissues between muscle fibers and along fascial planes and tendon sheaths. Moderate generalized osteoporosis results from progressive decalcification. In severe cases there may be pyrexia, progressive emaciation, muscle atrophy and joint stiffness. The subcutaneous nodules consisting of calcium phosphate and carbonate may gradually soften, the overlying skin ulcerate and secondary infection ensue. Death may result from sepsis.

**Treatment**—Recent investigations indicate that parathyroid hypertrophy and hyperplasia may result from vitamin D deficiency. Palliative treatment predicated upon this hypothesis comprises an abundance of ultra violet irradiation either by the sun's rays or lamps and a diet high in calcium salts. Viosterol and cod or halibut liver oil or their concentrates are recommended adjuvants. The therapeutic value of large doses of calcium (30 to 40 grains daily) is questionable. Parathormone administered intramuscularly in doses of 0.5 to 1 cc every ten days may augment calcium metabolism. Surgical therapy is generally limited to the evacuation and sterilization of superficial abscesses. Partial parathyroidectomy is indicated when a tumefaction is palpable in the region of the parathyroids or as a last resort in intractable and progressive cases.

## HEMIHYPERTROPHY

### (HEMIMACROSOMIA)

Hemihypertrophy is a rare developmental condition of unknown origin in which there occurs an asymmetrical enlargement of one half of the body or a portion thereof. The bones, muscles and blood vessels all share in the hypertrophy. Occurring in the arm, hand, leg or foot of the female, the affected part often assumes masculine characteristics, thickened derma, excessive hair, accelerated nail growth and increased muscular development. Callouses and nevi are rather commonly associated.

**Treatment**—There is no known method of arresting the process which ceases with full skeletal development. Corrective shoes may equalize a disproportion in leg length. One or more giant fingers or toes may be amputated to establish function of the part and in rare instances the size of an extremity may be reduced through plastic surgery. (Fig. 30)



FIG. 29 — Hemihypertrophy of the head and face



FIG. 30 — Hemihypertrophy of right upper extremity. Three giant digits and stail in infancy. Note excess of hair. (Case of Dr. J. C. Williams.)

## CHAPTER VII

### INDICATIONS OF BONE

The intimate connection between periosteum, cortex, cancellous tissue and bone-marrow precludes the possibility of infection being confined to a single structural element.

**Anatomy** —The Haversian systems constitute the structural units of bone. Each consists of a central channel, the Haversian canal, surrounded by superimposed layers of calcified connective tissue termed lamellæ. The lamellæ are composed of a finely reticulated structure, the matrix between the fibers being calcified (bone matrix). Between the lamellæ and arranged concentrically are numerous minute spaces, the lacunæ. These are connected with each other and with the central Haversian canal by numerous minute channels termed canaliculi. (Fig. 30.)

**Haversian Canals** —The Haversian canals are about  $\frac{1}{8}$  inch in diameter and run parallel to the long axis of the bone for short distances, then branch and communicate with neighboring canals. They contain blood vessels, nerve fibers, lymphatics and connective tissue. Through a system of intercommunication the Haversian canals open either on the cortical surface or into the medullary cavity. Whereas the lamellæ and lacunal spaces are arranged concentrically, the canaliculi radiate outward from the Haversian canals like the spokes of a wheel, passing across one lamella to another. Thus every part of the Haversian system receives nourishment derived from the Haversian canal vessels.

**Haversian System** —The Haversian system exhibits the form of a short cylinder, the Haversian canal running through its center. The cylinders are moulded into solid masses to form compact bone and into thin plates to form cancellous bone. In the latter framework Haversian canals are absent and the canaliculi open into the medullary spaces which perform the function of the canals.

**Bone Cells** —Each lacunal space is occupied by a bone cell (bone corpuscle). These are flattened, nucleated, branching cells homologous with connective tissue cells. Their branching processes pass through the canaliculi and in young growing bones there is a communication thereby with neighboring cells in other lacunæ and also with similar cells found in the Haversian canals. (Refer to Development of Bone.)

**Classification of Bones** —Bones are classified as long bones, irregular bones and flat bones. A long bone is a tubular structure



whose length exceeds its breadth and consists of a shaft (diaphysis) and two extremities (epiphyses). Irregular bones as described by the name may be considered as long bones whose ends have been pushed together thus obliterating the medullary cavity. Flat bones are often irregular in shape and consist of two plates of compact bone separated by a small amount of cancellous tissue.



**Periosteum**—The periosteum is a highly vascular membrane which envelops the entire bone except at its articular ends. It is attached to the cortex by fibrous connections (Sharpey's fibers) and by vascular and lymphatic vessels which pass through the Haversian canals. At the epiphyseal ends the periosteum dips into the epiphyseal line and becomes densely adherent to the epiphyseal cartilage, also it is not continuous with the periosteum which covers the epiphysis (Fig 32). Where tendons are attached to bone they become incorporated with the periosteum and the fibers permeate the cortex for anchorage. The periosteum is composed of two layers, an outer of connective tissue and an inner of densely arranged elastic fibers (this comprises several layers). Between the periosteum and cortical bone there is a layer of scattered osteoblastic cells which are extremely important in bone growth and repair. The internal periosteum (endosteum) is a highly vascular areolar membrane and contains abundant osteoblastic tissue. It lines the medullary cavity and acts as an envelope for the marrow.

**Cortex**—This dense structure is composed of lamellae arranged (1) parallel with the periphery, (2) concentrically about the Haversian canals and (3) irregularly in the spaces between the Haversian systems. The compact bone is thickest at the center of the shaft ( $\frac{1}{8}$  to  $\frac{1}{2}$  inch) and tapers toward the epiphyseal line to paper thinness. This is the weak point of the cortical architecture and represents the site at which early infection of the cancellous tissue may break through to the subperiosteal space (Fig 33). Blood vessels and lymphatics pass from the periosteum to the medulla via the Haversian canals.

**Cancellous Bone**—This is a much looser mesh structure than compact bone and the trabeculae are arranged in such manner as

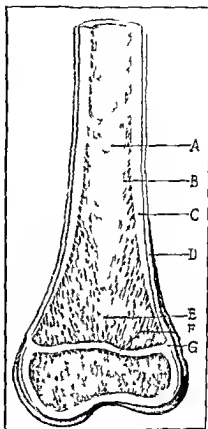


FIG 32—Section of lower end of a child's femur. A Marrow B endosteum C compact bone D periosteum E cancellous tissue F epiphyseal cartilage G insertion of periosteum into epiphyseal cartilage

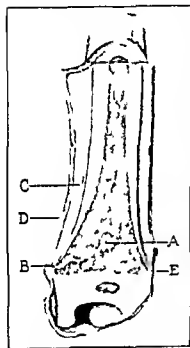
will best withstand stress and strain. The cancellous tissue is thinnest in the mid third of the shaft and increases in amount toward the ends of the bone being most abundant between the ends of the medullary cavity and the epiphyses.

**Medullary Cavity** — The central canal of long bones is lined by a highly vascular membrane, the endosteum or internal periosteum, and contains bone-marrow.

**Bone-marrow** This not only fills the medullary cavity of the shafts of long bones and the spaces in the cancellous tissue but also

extends into the larger Haversian canals. It consists of a matrix of fibrous tissue supporting numerous blood vessels, fat and marrow cells. The latter resembling lymphoid corpuscles contain small pinkish cells (erythroblasts). In the epiphyses of long bones, in the sternum, ribs, vertebra, flat and short bones and the cranial diploe, the predominance of erythroblasts imparts a red color to the marrow.

**Epiphyseal Cartilage** — In a growing bone the diaphysis is separated from the epiphysis by a layer of cartilage cells termed the epiphyseal plate or conjugal cartilage. With full skeletal growth the cartilage cells become ossified and the diaphysis and epiphyses fuse. Forcible strain may produce separation of an epiphysis; the line of cleavage oc-



**Nutrient Arterial System**—The nutrient artery penetrates the diaphysis at either side of the mid-point of the cortex and divides into two main branches directed toward each end. These continue to branch, forming a rich network in the medullary cavity. In adult bone the terminal vessels anastomose with branches from both the periosteal and irregular systems. In young bones retaining the epiphyseal cartilage, the terminal vessels become end vessels as they approach the cartilage plates and communicate by loops with the return venous network. Thus on the diaphyseal side of the epiphyseal cartilage there is a narrow, relatively avascular zone where the blood stream is definitely slowed. This is a common site of bacterial invasion in acute hematogenous osteomyelitis (Fig 33.)

**Periosteal Vascular System**—The inner layer of the periosteum has an abundant vascular plexus. Numerous vessels pass through the small apertures in the cortex and, proceeding through the canals of the cortical substance, reach the cancellous tissue and ultimately anastomose with the nutrient arterial network. It should be emphasized that the major vascular supply of the superficial layers of the cortex is of periosteal origin and that necrosis of the outer layers of the cortex may result if the periosteum is elevated or its vessels are thrombosed.

**Irregular Vascular System**—These vessels penetrate the larger and smaller apertures in the ends of long bones and supply the epiphyses and their bony eminences. In growing bones, the epiphyseal cartilage segregates this system from the other two, after ossification occurs, free anastomosis becomes established.

The medullary cavity is thus supplied by the nutrient artery, the cancellous tissue and diaphyseal cortex, by both the nutrient artery and the anastomatic plexus of the periosteal system, the epiphyses in growing bones by the irregular system, and after completion of ossification by the irregular system and the anastomatic network of the nutrient artery. Veins generally accompany the arteries. In the flat cranial bones the veins attain large size and are contained within the lamellated walls of the diploe.

**Lymphatics**—Klein has traced the periosteal lymphatics into the Haversian canals and Crankshank, into the bone substance.

**Bone Development**—Bones develop in either cartilage or membrane from preliminary models laid down during fetal life, the long and irregular bones being performed in the former and the flat skull bones in the latter. Ossification begins in localized areas termed ossification centers. Whereas some irregular bones have only a solitary center, the long bones contain three, one being centrally placed, the others at each end. The central ossification center develops more rapidly than the distal ones. A typical long bone in a child consists of an osseous shaft (diaphysis) and two osseous ends (epiphyses), separated by thin epiphyseal or conjugal cartilages,

or plates. These persist a variable length of time in different bones until final ossification occurs with full skeletal maturity (twenty-one to twenty three years).

**Bone Growth** — Normal growth of bone occurs through the agency of certain large cells homologous with connective tissue cells termed osteoblasts. As the pericellular ground substance is replaced by a calcareous matrix the osteoblasts become enclosed in lacunal spaces between the lamellae of the Haversian systems and are termed bone cells. Their bone-forming potential then becomes dormant and their function is associated with the nourishment and viability of the bone. Osteoblastic activity is greatest in the regions of the epiphyseal cartilages and subjacent to the inner layer of the periosteum and is definitely less marked in the endosteal tissue between the marrow and cancellous bone.

Growth in the longitudinal axis occurs from the epiphyseal cartilages at each end of a long bone. This growth rate however is unequal; it is more rapid in the upper epiphysis of the humerus and lower epiphyses of the radius and ulna and in the lower epiphysis of the femur and upper epiphyses of the tibia and fibula. Growth in the transverse axis results from a combination of bone accretion and absorption. Concurrently with the laying down of new lamellae between the periosteum and cortex the medullary cavity is enlarged through the absorption of cancellous tissue adjacent to the endosteum. The accretion is produced by the periosteal osteoblasts and absorption by the osteoclasts which lie between the endosteum and bordering cancellous tissue. These scavenger like osteoclasts are multilocular cells of the giant type and lie in the outskirts of the marrow in semilunar spaces termed Howship's lacunae. Their function is to decalcify the intercellular matrix the lime content being removed in the blood serum.

**Bone Repair** Reporative processes are greatest in young bones gradually diminish with advancing years and become feeble in the aged. Bone tissue destroyed through infection is not regenerated or only feebly so. Small particles of necrotic bone can be absorbed by the osteoclasts (molecular erosion). Large segments however are beyond the power of osteoblastic absorption. Maximum reparative processes occur both in the union of fractures and of separated segments resulting from osteomyelitis. The callous formation results from a combination of reparative agencies: (1) Periosteal and endosteal osteoblasts, (2) adult bone cells exposed in the line of fracture which when released from their lacunae lose their dormancy and resume osteoblastic activity and (3) homologous connective tissue cells which appear in the extravasated blood.

In most cases of osteomyelitis the involucrum is formed by the periosteal osteoblasts as the infection generally destroys all other osteoblastic tissue. Masses of new bone may thus be built down

between the periosteum and shaft. When in rare instances the periosteal osteoblasts are also destroyed new bone formation is wanting. Healing then occurs by fibrous union and if a break in bone continuity has occurred a false point of motion obtains.

### ACUTE HEMATOGENOUS OSTEOMYELITIS

Acute hematogenous osteomyelitis is an acute metastatic or subsidiary focus of infection in the bone the result of blood borne bacteria demonstrable or undemonstrable from a primary focal infection of the body surface skin alimentary canal respiratory genito-urinary or otologic tract.

**Etiology**—The infective agents are generally the staphylococcus or streptococcus less frequently the pneumococcus influenza or typhoid bacillus and rarely the colon or paratyphoid. Approximately 80 per cent of the cases are due to the *Staphylococcus aureus* or *albus* the primary nidus being some infection of the skin surface furunculosis boils carbuncles infected wounds or rarely umbilical sepsis. The streptococcus usually enters the blood stream from infections of the nasopharynx tonsils teeth or sinuses. Mixed infections (symplicosis) with more than one organism occur rarely except after sinus formation.

**Age**—Although occurring at all ages acute hematogenous osteomyelitis is preeminently a disease of childhood and most cases occur between the ages of two and fifteen years. Infection is definitely less common after bony fusion of the epiphyses. (Adults are more likely to develop arthritis in the presence of a blood-stream infection.)

**Sex**—Boys are affected more frequently. They play harder than girls and are more subject to trauma scratches and abrasions also to acne and furunculosis.

**Trauma**—In a considerable minority of cases trauma bears a definite causative relationship to the initial area of infection. It has been amply demonstrated that slight trauma may cause fracture of the delicate bone trabeculae resulting in hemorrhage and interference with nutrition. A sprain may produce bleeding through partial separation of an epiphysis. (Refer to Anatomy.) Such areas of lowered vitality predispose to the localization of blood borne organisms. In unusual instances acute osteomyelitis may develop in simple fractures.

**Sites of Infection**—The common sites of involvement in order of frequency appear to be the upper end of the tibia lower end of the femur and upper end of the humerus also the lower end of the humerus tibia fibula or radius upper end of the femur and the iliac crest (Wilensky). At times multiple foci develop in different bones.

**Bacteriemia.**—Clinical cases present three types: (1) Acute osteomyelitis without bacteriemia. In this group the sequence of events consists of a primary focal infection from temporary bacteriemia, the development of a fixation point in the bone, and disappearance of the bacteriemia. (2) Acute osteomyelitis with demonstrable living bacteria in the blood stream. In these cases there is an infected thrombo-embolus formation which continues to feed the blood stream viable organisms. Following appropriate surgery the bacteriemia may disappear and recovery follow. (3) Cases of profound general infection. Highly virulent organisms multiply in the blood stream and the patient becomes overwhelmed by the toxemia. The rôle of the infected thrombo-embolus is negligible; the bacteria multiply in the blood stream through the patient's lack of resistance and the inextensive area of osteomyelitis is an inconsequential factor in a multiplicity of foci. Septic endocarditis is a common complication. Death occurs early from profound toxemia.

**Pathogenesis.**—Osteomyelitis results from the hematogenous deposition of isolated organisms, clumps of organisms, or fragments of infected clots which are derived from some primary surface nidus. The bacteria enter the bone through either the nutrient artery or the periosteal vascular system and lodge at various sites of the circulation (fixation points). Thrombo-embolism is most likely to occur where there is retardation of the blood stream. (At times this is associated with accidents in the local bone circulation.) In the juxta-epiphyseal region (metaphysis) the small end-vessels terminate in large capillary loops and the resulting stasis favors the development of thrombo-embolism.

The various sites of election for fixation points (foci of osteomyelitis) in order of frequency are: (1) In the periosteal vascular plexus, (2) in the superficial Haversian canals of the cortex; (3) in the metaphysis, (4) in the main trunk of the nutrient artery; and (5) in one of the major or minor branches of the nutrient artery (Wilensky).

**Vascular Thrombosis.**—The dominant element in the development of osteomyelitis is vascular thrombosis. Depending upon the size of the vessel primarily involved, the subsequent spread of the thrombosis and the collateral circulation, small or large areas of bone become devitalized. Thus, if the thrombo-embolus involves the terminal vessels in the metaphysis and no retrograde thrombosis occurs, the damaged area will be small and circumscribed; if on the other hand the main nutrient artery is occluded, the entire diaphysis will become devitalized (Fig. 31). When infection persists in the clotted area, the thrombus may spread distally to smaller vessels or retrogradely to larger tributaries. Extensive thrombophlebitis is usually accompanied by a demonstrable bacteriemia. Many of the so-called recrudescences and recurrences may be

explained on the basis of spreading thrombosis. This may not only occur before operation but also as the result of operative interference, or at any subsequent period.

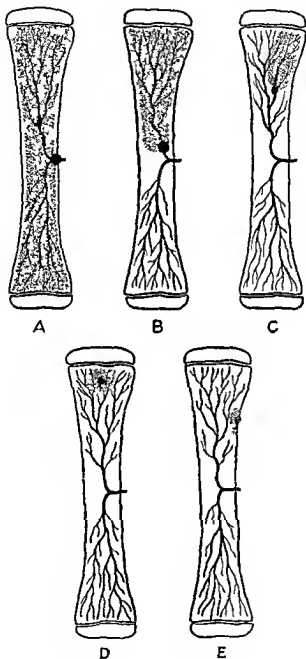


FIG. 34.—Various types of thrombo-embolism.



**Abscess Formation**—There are three clinical varieties—subperiosteal, intraosseous, and medullary abscess. Subperiosteal abscesses are the most common. They may result from (1) suppuration of a focal process in the periosteal vascular network (the adjacent cortical bone is not necessarily involved), (2) a small cortical abscess may rupture into the subperiosteal space, (3) a medullary abscess may perforate into the subperiosteal space, and (4) necrotic and sequestered bone under the periosteum may produce suppuration. The abscess may be small or the purulent material may be sufficiently abundant to elevate the entire periosteum from the shaft, being limited at either end by the periosteal attachment to the epiphyseal cartilages. Unless relieved the periosteum usually perforates and the soft parts become invaded. Suppuration in the irregular bones is prone to involve neighboring joints.

Intraosseous abscesses result from infection of the vascular network of either the cortex or the cancellous tissue. Whereas the former occurs uncommonly, the latter is a frequent sequel of infection in the end-capillary loops of the nutrient artery in the metaphysis.

Medullary abscesses result from infection in the fine vascular network of the smaller branches of the nutrient artery supplying the marrow. They may be small and resolve or be extensive and

development of new bone. (The role of dead bone simulates that of a bone graft.)

**Absorption of Dead Bone**—This is due to lacunal erosion by the osteoclasts. At the boundaries between living and dead bone small mononuclear cells develop into large polymorphonuclear cells termed osteoclasts. These cells apparently originate from progenitors that may develop into either osteoblasts or osteoclasts. The cells lie in absorption lacunae (Howship's lacunae) and by a lytic process produce bone erosion and absorption. (The jagged edges occurring in dead bone result from irregular erosion.) Ultimately the osteoclasts revert to osteoblasts or bone cells after their osteoclastic function has been fulfilled.

When necrosis occurs in the process of a long bone which enters into the formation of a joint, complete absorption of the dead bone almost always occurs and the bone is never replaced. In necrosis of the head of the femur follows thrombo-embolism of the vessels in the ligamentum teres. Osteomyelitis of the irregular carpal and tarsal bones also usually eventuates in complete absorption of the necrosed bone. Infection of the cranial bones causes a loss of bone tissue either through absorption or sequestration. Osteoblastic activity is rendered inert by the infection and the bone defect is permanent.

**Reparative Processes**—Defects in bone tissue whether due to molecular absorption, sequestration or operative osteotomy are very seldom replaced by new bone formation. Minimum surface defects are replaced by firm scar tissue which is supported by involucrum formation. Larger defects are filled in with granulation tissue which later becomes a firm fibrous scar and bone tissue of any degree does not grow out to fill the defect. In very young children, however, the amount of new bone tissue thrown out combined with the osteoblastic activity of growth often replaces the defect more or less completely.

Excavation of the entire diaphysis of a long bone may be due either to obliteration of the nutrient artery alone or of both the nutrient artery and the periosteal vascular system. In the latter case the shaft defect is replaced by fibrous tissue (fibrous union) with a resulting false point of motion (pseudo-arthritis). When the periosteal circulation is undamaged periosteal bone formation is greatly stimulated and bone continuity is preserved through the development of a massive involucrum. Articular extremities and processes, also carpal and tarsal bones which become absorbed are never replaced.

Bone repair is produced by the osteoblasts and certain genetically related cells of equivalent potential—osteoblastic layers of the periosteum, bone cells and epithelial like cells which line the marrow cavity (endosteum) and Haversian canals. Marrow cells

bones and typical gross pathologic patterns can be distinguished in most cases (Fig 34)

1 Lesions in the nutrient artery producing involvement of the entire diaphysis (Fig 35)

2 Lesions in the primary branch of the nutrient artery producing involvement of one-half of the diaphysis (Fig 36)

3 Lesions in branches of the nutrient artery producing involvement of larger or

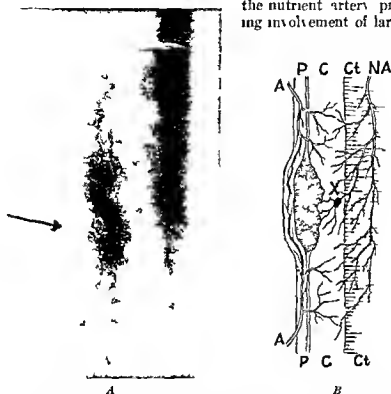


FIG 37 A Cortical abscess from thrombo-embolism of the cortical network of the nutrient artery B Diagrammatic representation of same P C and Ct periosteum cortex and cancellous tissue respectively A periosteal vascular network A branch of nutrient artery with thrombo-embolism at X

smaller segments of the diaphysis (infarcts) depending upon the size of the occluded branch (Fig 37)

4 Lesions in the terminal vessels of the nutrient artery producing involvement of the metaphysis (Fig 38)

5 Lesions in the periosteal vascular network producing involvement of the corresponding portion of the periosteum with or without cortical invasion (Fig 39)

Most clinical cases accord in general with the foregoing types. Although at times the demarcation is less marked, careful study will usually reveal a rather definite pathologic pattern. The end results

of the osteomyelitis also accord with the classification the area of molecular erosion or sequestration corresponding to the zone of infarction

**Symptomatology** In approximately 90 per cent of cases the lesions occur in long bones. The symptoms are variable depending upon the virulence of the infecting organism. In fulminating cases the clinical picture is one of profound general sepsis—sudden onset repeated chills sustained hyperpyrexia rapid feeble pulse dry



FIG. 38. Osteomyelitis in metaphysis with diffusing abscess formation.

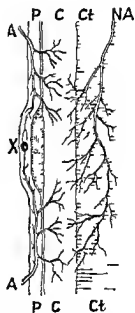
tongue and scanty urine containing albumin and casts. The patients are severely prostrated, often delirious and succumb in forty-eight to seventy-two hours. The local bone lesion is seldom demonstrable for there are few or no focal signs; the fixation point plays no role in the pathology which is one of a multiplicity of foci. Bacteremia is aggressive and the blood cultures commonly exhibit staphylococci and less often streptococci. The diagnosis of osteomyelitis is generally made at autopsy. In less severe types the patients survive a few days longer and die of pneumonia, metastatic

meningitis purulent effusions into the serous cavities or acute nephritis. Septic endocarditis is a usual complication.

In the average case there is generally an antecedent history of boils, furunculosis, tonsillitis, respiratory infection or perhaps a febrile attack with intestinal symptoms (colon bacillus infection). Such history is important in evaluating the probable type of infection. Frequently there is also a history of trauma, especially of a sprain. Although a slight epiphyseal injury may appear inconsequential, the attendant hemorrhage and tissue damage definitely predispose to bacterial invasion (Fig. 36). Following an incubation



A



B

FIG. 39. A Subperiosteal abscess due to thrombo-embolism of the periosteal vascular network. B Diagrammatic representation of same. P, C, and Ct, periosteum, cortex and cancellous tissue respectively. A, periosteal vascular network with thrombo-embolism at A. NA, nutrient artery.

tion period of a day to several weeks prodromal symptoms of malaise, headache and anorexia develop. This premonitory period is usually brief and is followed by a chill, hyperpyrexia and the development of the dominant syndrome of pain and stiffness near a joint.

Pain is due to intramedullary or intraperiosteal tension. It is of severe, constant, throbbing, boring type without remission and worse at night. It is also aggravated by heat. Stiffness is referred to the neighboring joint and movement aggravates the pain. The

affected part is rigidly guarded against motion or jarring. The focal tenderness is definitely limited to the zone of bone involvement; joint involvement can be excluded by careful examination. The temperature usually remains high with corresponding elevation of pulse and respiration.

**Tenderness** is definitely localized. In metaphyseal infections the spread of tenderness is toward the center of the bone, whereas in diaphyseal lesions it is distal in both directions from the focal point. It cannot be overemphasized that pain and focal tenderness are the only objective findings in the first twenty-four to forty-eight hours. After a few days or a longer period local edema may develop, followed by redness. Edema of the parts distal to the infection may result from the focal thrombophlebitis.

Thus in the average case the local lesion plays a dominant role. The pain lessens with rupture of the periosteum and neighboring muscles and fascial planes become infected as the pus travels along the lines of least resistance. At any time in the course of the disease but especially in the early stage exacerbations may occur from the development of new foci, and new fixation points may also become established through spread of the thrombo-embolus. Blood cultures are sterile in the majority of cases. Bacteriemia connotes extensive thrombophlebitis.

**Multiplicity of foci** occur in most cases and are a marked feature of the pathology. There is a wide variation in the intensity of the exacerbations and slight ones are often overlooked. After twenty-four to forty-eight hours it is not unusual for another bone to become infected.

**Fever** is of several varieties. In fulminating cases it may reach  $106^{\circ}$  or  $107^{\circ}$  F. and remain constantly high until death. In the average case the temperature is maintained at  $103^{\circ}$  to  $104^{\circ}$  F. and the patients may succumb to toxemia unless the pus is evacuated. Following adequate drainage the temperature generally subsides. Intermittent fever with wide daily swings of  $5^{\circ}$  to  $7^{\circ}$  denotes a generalized infection. This is not necessarily fatal, however, unless complications develop. The irregular exacerbations so common in the progress of the disease are generally due either to insufficient drainage or to the development of fresh foci.

**Blood.** In the acute stage the blood exhibits a leukocytosis of 15,000 to 30,000 with neutrophilic polymorphonuclears of 85 to 95 per cent; the lymphocytes are greatly reduced and the myelocytes increased. With satisfactory progress there is a gradual return to normal. Progressive anemia results from destruction of the erythrocytes and the red cells and hemoglobin may be reduced to even 30 per cent.

**Lymphatics.** The regional lymph nodes may be enlarged and tender.

**Clinical Course**—As the disease progresses to the healing or subacute stage necrotic bone is either molecularly eroded or sequestered and the bone defect is repaired by granulation tissue and involucrum formation. The temperature gradually becomes normal in five to eight weeks which is the average period required for the separation of dead from viable bone. During this period the soft parts and osteotomy cavity fill with granulations and one or more sinuses generally develop. If all the necrotic bone is removed by molecular erosion the sinuses will close. This occurs decidedly more often in infants than in older children. Persisting sinuses commonly discharge a thin watery pus indicative of residual sequestra. The latter when small may be extruded through the clover. Before this occurs the ostium becomes inflamed and the discharge fetid. Larger sequestra require operative removal. Exacerbations are common at this stage. They may result from insufficient drainage or the development of fresh foci of infection in either the bone or involucrum. Recurrences may also occur years after healing from residual foci.

**Differential Diagnosis**—*Acute rheumatic fever* is the most common erroneous diagnosis through mistaking the lesion to be intra-articular. Rheumatic fever is generally polyarticular the patient is less prostrated the pain is more remitting leukocytosis seldom exceeds 15,000 suppuration does not occur and blood cultures and radiographs are negative. *Cellulitis* of the deep structures near a bone may be confusing and at times only the progress of the disease will differentiate it from bone infection. Scurvy produces subperiosteal hemorrhage as well as bleeding gums. Fever is slight and there is a history of vitamin deficiency. Although epiphyseal separation may occur in both osteoporosis is absent in scurvy. *Septic arthritis*. In young infants and very sick children it is often impossible to elicit the exact location of the tenderness. If bacteremia occurs the presence of staphylococcus favors a bone lesion whereas streptococcus or pneumococcus suggests joint invasion. *Tuberculosis*. There is seldom any difficulty in differentiating tuberculosis. The insidious onset slight fever absence of leukocytosis and radiographic evidence of epiphyseal destruction with joint involvement exclude osteomyelitis. *Syphilis*. A subperiosteal deposit of hard bone occurs commonly in lues. However the new bone laid down in osteomyelitis is definitely more excessive and irregular. Syphilitic epiphysitis though painful causes only slight temperature and the absence of hyperpyrexia and leukocytosis is associated with a positive Wassermann reaction. Other evidences of hereditary lues are also usually exhibited. (Refer to Syphilis.) *Myositis ossificans traumatica* is a slow painless development of bone in an old blood clot. The new bone is deposited in lamellations parallel to the long axis of the bone and muscles and the

process is unaccompanied by fever or leukocytosis (Refer to *Mycosis Ossificans*)

**Roentgenology**—It cannot be too strongly emphasized that in the early stages of acute osteomyelitis the roentgen ray findings are entirely negative. The earliest demonstrable evidence occurs about the eighth day—linear separation of the periosteum from the cortex over the site of infection (Fig 40). This periosteal elevation may be slight or extend the entire length of the diaphysis. At this period the bone texture appears normal. The earliest evidence

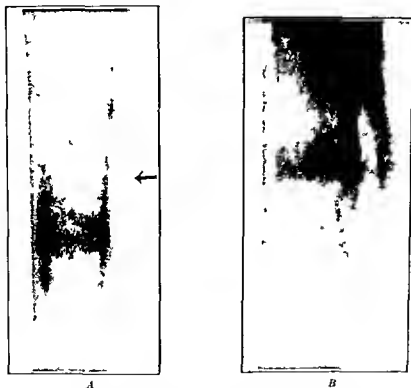


FIG 40 A Early cortical osteomyelitis B at a later date

of actual bone change occurs from the tenth to the fourteenth day, or later—rarefaction of the bone due to the absorption of lime salts. This may be exhibited either as a single area of increased radiability or as several areas producing a mottled appearance. In the early stages of the disease, the foregoing are the only demonstrable roentgenologic findings.

In moderately advanced stages the pathologic processes exhibit a variety of findings—bone destruction, sequestration and involucrum formation. The dominant factor is bone destruction with a mini-



num of new bone formation (In chronic osteomyelitis the reverse occurs) Destruction of the endosteum produces areas of increased radiability whereas that of the cortex results in bone destruction and sequestration periosteal stripping and the laying down of new irregular bone or involucrum The full extent of destruction is not evidenced for several weeks and serial roentgen rays taken every ten days may exhibit an apparent spread of the disease Whereas the magnitude of destruction occurs early the nature of the pathologic processes requires time to produce roentgenologic demonstration

In advanced cases certain anatomic types can usually be established from the roentgen rays

1 *Periosteal Foci*—A subperiosteal abscess resulting from invasion of the periosteal vascular network is represented in Fig 39 A The lesion and its genesis are diagrammatically represented in Fig 39 B In this type the cortex is not involved and evacuation of the pus will result in healing

2 *Cortical Foci*—The pathology results from a thrombo embolism of the cortical network of the nutrient artery Abundant collateral network limits the area to a small necrotic segment of cortex A small subperiosteal abscess is a common accompaniment Sequestration with mild involucrum formation results (Fig 37) If the periosteum is extensively elevated a thick involucrum develops

3 *Nutrient Artery Foci* Necrosis of the entire diaphysis occurs from thrombo-embolism of the main nutrient artery (Fig 35) The periosteum becomes hypervascular and lays down a thick irregular layer of new bone (involucrum) which surrounds the sequestered diaphysis and gives support to the bone

4 *Primary Nutrient Branch Foci* The pathology is represented in Figs 34 B and 36 Practically one-half of the diaphysis is involved and this occurs through the entire thickness of the shaft

5 *Subsidiary Nutrient Branch Foci*—A corresponding smaller segment becomes involved depending upon the collateral circulation The area does not include the entire thickness of the diaphysis (Fig 34 C)

6 *Terminal Nutrient Foci* The metaphyseal lesion exhibited in Fig 38 is the result of thrombosis of the terminal nutrient vessels Such infection may spread in several channels In mild infections the zone may become surrounded by granulation tissue the organisms may die and a collection of sterile pus may persist within the bony inclosure This is the genesis of the chronic bone abscesses first described by Brodie

The foregoing anatomic types are generally demonstrable during the third and fourth weeks of the disease The roentgen pattern may be complicated due to the development of several different foci or to an irregular spread of the thrombus Cases in which both

the periosteal and nutrient vessels are involved exhibit a solution of shaft continuity. Osteotomy may further complicate the roentgenogram. After the process has continued for months (chronic osteomyelitis) there is an excess of bone reproduction with smaller areas of destruction. The shaft may become greatly thickened and irregular and the medullary cavity practically obliterated. Irregular shaped cavities may be the result of bone deficiency or operative interference.



FIG. 41 Epiphysitis and abscess in diaphysis

lar shaped cavities may be the result of bone deficiency or operative interference.

**Acute Epiphysitis** Acute epiphysitis is a variety of osteomyelitis in which infection occurs in the epiphysis (Fig. 41). Infants and young children are chiefly affected. The etiology and pathogenesis are the same as those of diaphyseal infections. (Trauma plays an important role in many cases.) Depending upon the size of the vessel or vessels involved by the thrombo-arteritis or thrombophlebitis the degree of bone and cartilage destruction varies from small necrotic areas to total epiphysal destruction i. e. necrosis of the head of the femur results from thrombo-embolism of the vessels in the ligamentum teres.

Acute epiphysitis may be entirely intracapsular partially so or extracapsular. When the area of osteomyelitis is intracapsular ankylosis usually results.

and dominates the clinical picture. As previously stated intracapsular necrotic bone and cartilage are not replaced by new osseous formation. If the epiphysal cartilage is involved and this occurs chiefly in young bones longitudinal growth may become impaired.

**Acute Bone Abscess** — Acute bone abscesses commonly occur with osteomyelitis. The purulent material may accumulate in either the cancellous mesh or in the medullary cavity often both struc-

tures are involved. In medullary abscess the entire marrow cavity becomes rapidly invaded resulting in extensive bone necrosis and sequestration. The subsequent repair is sluggish (Fig 35).

The clinical course differs in no way from that of acute osteomyelitis: chills, sustained hyperpyrexia, focal tenderness, leukocytosis, etc. The purulent material frequently perforates the cortex producing a subperiosteal abscess which subsequently ruptures into the soft parts. Adequate drainage results in subsidence of the constitutional symptoms. Secondary abscesses may develop from either pocketing or the development of secondary foci. In certain instances of mild infections the abscess may become localized in the medulla and produce a chronic abscess. When such processes are multiple the bone presents a honeycomb appearance.

**Chronic Bone Abscess** — The pathology may result from either an acute abscess or the residuum of focal necrosis in the metaphysis following thrombo-embolism of the terminal nutrient vessels. The latter variety comprises the chronic type of abscess described by Brodie in 1830. The abscess is small, often oval in shape and is surrounded by a zone of fibrosis. Hypertrophy of the overlying cortex and periosteum may accompany the condition (Fig 42). Such lesions may be asymptomatic for years. The usual complaint is mild bone ache which is worse at night; acute exacerbations rarely occur. The pathology is readily demonstrable roentgenologically. Evacuation of the pus usually eventuates in prompt healing. Cultures are generally sterile and when positive the organism is almost always the staphylococcus.

**Complications of Acute Osteomyelitis** — Abscess is the most common complication (25 to 50 per cent of cases). It may be intra-



FIG 42 — Brodie's abscess. Hypertrophy of the overlying cortex and periosteum.

osseous, subperiosteal or intramural. *Joint complications* are less frequent and occur in 5 to 10 per cent of the cases, effusion being more common than suppuration. *Growth disturbance* following osteomyelitis consists of either lengthening or shortening of the bone, depending upon whether the diaphysis or epiphysis is involved. Lengthening may be produced by diaphyseal disease from hyperemia and overstimulation of the growing epiphysis, and diminution or cessation of growth may result from either infection of the epiphyseal cartilage or detachment of the epiphysis. Visceral complications are unusual except in cases of severe bacteriemia. Amyloid degeneration is rare.

**Prognosis**—Whereas the statistics of different authors exhibit wide variations, the average mortality rate appears to be about 10 per cent. Initial sepsis is the dominant lethal factor and most deaths occur in the first week. Skull cases are particularly serious due to the danger of intracranial complications, and spinal lesions may eventuate in meningitis. Severe pyarthrosis is also a grave complication. Insofar as the local lesion is concerned, osteomyelitis of the long bones offers the best prognosis. The severity of the bacteriemia may be roughly evaluated by the number of colonies; a rapid increase in number is ominous, likewise, endocarditis, hemorrhages into the skin and icterus. Recovery does not always occur in "sterile culture" cases as a late bacteriemia or fatal complication may develop.

Permanent cures probably obtain in 50 to 75 per cent of cases and these occur most often in young children. Recurrence, however, may develop after many years of apparent cure. Disturbance of bone growth is evidenced in a small percentage of cases. Pyarthrosis generally eventuates in ankylosis, amputation, however, is seldom necessary.

## TREATMENT OF ACUTE HEMATOGENOUS OSTEOMYELITIS

This comprises the treatment of (1) the general infection (bacteriemia) and (2) the local lesion.

Repeated blood cultures should always be taken because a single or several negative cultures may be followed by positive findings. In the presence of bacteriemia, repeated cultures give a rough evaluation of the progress of the disease; an increase in the number of colonies connotes an aggressive bacteriemia and a decrease indicates subsidence.

Clinically the cases comprise three groups: (1) Those with sterile blood cultures; (2) positive cultures in fulminating types, and (3) positive cultures in the average case. In Groups 1 and 2 there is no indication for immediate operation; in the former the process is well fortified by Nature and there is a definite advantage in waiting,

in the latter the focal process is an inconsequential part of a multiplicity of foci. In Group 3 operation is only indicated in certain cases in which the bacteriemia is progressively increasing and there is reason to believe it is due to spreading thrombophlebitis in the bone. In such cases radical osteotomy should be performed to remove all foci.

**Indications for Operation**—In the absence of bacteriemia and in the average case accompanied by mild infection operation is contraindicated *unless suppuration develops* within the bone beneath the periosteum or in the soft parts.

Many surgeons believe that immediate operation is indicated in all cases of acute osteomyelitis. The hypothesis is predicated upon the concept of bacteriemia developing from the bone lesion. This however only occurs in unusual cases of spreading thrombophlebitis. Moreover an ill timed operation may actually precipitate bacteriemia in a previously sterile blood stream.

In the absence of pus delay in operation has many advantages: uninvolved bone tissue is conserved, the thrombophlebitis is not spread by operative trauma, small sequestra especially intracapsular segments from acute epiphysitis are absorbed, and a certain number of cases recover without operation. *Absolute immobilization of the part is imperative* through the aid of a splint of moulded plaster. When joints are involved suitable traction should also be provided. Except in knee-joint infections pyarthroses are best treated by conservative measures unless the capsule ruptures and the soft parts become invaded.

The cardinal indication for operation is the presence of purulent material. This should be evacuated by the simplest procedures with minimum damage to the bone tissue. A subperiosteal abscess should be treated by incision of the periosteum and drainage (soft rubber tubular drainage). A medullary abscess when localized may be evacuated through drill holes, extensive medullary suppuration however requires adequate osteotomy for drainage. In general osteotomies are reserved for a later stage when sequestration and involucrum formation have occurred.

Following conservative operative treatment one of two conditions will obtain: (1) The lesion will heal or (2) necrotic bone will be sequestered in about six weeks. As previously stated (refer to Pathology) small sequestra may be removed by molecular erosion and larger segments may either become revascularized and incorporated with the bone or be spontaneously extruded. Large sequestra require operative removal (sequestrotomy).

**Sequestrotomy**—This procedure becomes indicated when the constitutional reaction has abated and serial roentgenograms indicate that the segment has separated and that revascularization is not occurring. The operation should be performed under general

anesthesia The wound is carefully sterilized by scrubbing with green soap and water followed by ether and the surface is then painted with iodine All granulation tissue is curetted from the sinuses The incision is planned as will best approach the diseased bone and as little involucrum as possible is removed to extract the



FIG. 43 —Residual cortical abscess in chronic osteomyelitis (Courtesy of Dr. E. C. Hansen)

*sequestrum en masse* The cavity is then packed with iodoform gauze covered with a sterile dressing and the extremity is immobilized Most cases progress in a sterile manner and the wound is accordingly undisturbed for two weeks The patient is then taken to the operating room and the dressing removed If the wound is clean secondary suture of the soft parts is performed and in many

instances permanent healing occurs. If infection develops the wound is promptly reopened and drained.

**Hygienic dietetic Measures**—These are highly important during convalescence. abundant fluids, high caloric diet, fresh air, sunshine or artificial ultra violet irradiation, hematinics, etc. Repeated blood transfusions are of great value in cases of severe secondary anemia. At times vaccines and bacteriophages are also helpful. Chemical sterilization of the blood stream with mercurochrome or gentian violet is not recommended.

## CHRONIC OSTEOMYELITIS

By common usage the term chronic osteomyelitis is applied to cases which do not heal following the usual regimen of treatment and pursue a chronic course. Persistence of the pathology may be due to one or a combination of several causes. (1) *Foreign bodies*. Segments of dead bone, either completely sequestered or incompletely revascularized, are the most common cause of chronicity. (2) *Retention of infected foci*. As previously mentioned, infected bone foci occur frequently (Fig. 43). In some instances temporary healing occurs followed by recrudescences. Foci of infection may also be retained in the involucrum or in the scar. (3) *Insufficient drainage*. Pocketing may occur in the bone or soft parts. (4) *Rigid walls* may prevent cicatrization. (5) *Joint involvement*. (6) *Poor general condition* of the patient. This important factor is often overlooked.

**Treatment**—This comprises both constitutional and local measures. Prolonged infection often so debilitates a patient that the physiologic processes of repair become exhausted. It cannot be too strongly emphasized that hygienic dietetic measures combined with heliotherapy and repeated blood transfusions are extremely valuable. Children appear to thrive best at the seashore.

**Local Treatment**—This is governed by the underlying pathology. In general it comprises: (1) The removal of all infectious foci in the bone, involucrum and scar. (2) sequestrotomy, and (3) a suitable toilette of the viable bone to promote permanent healing. A properly planned osteotomy should permit of free access to the pathology. After an adequate incision has been made in the soft parts, the periosteum is incised and elevated for about one-third of its diameter. This should be performed close to the cortex to conserve the cambial layer. A sizable osteotomy wedge is then removed (often the involucrum) and the cavity of the bone is carefully explored for granulation tissue (infection foci), sequestra and jagged bone particles, all of which should be meticulously removed. Bone sinuses should be explored and thoroughly opened. After all the evidences of infection and foreign bodies have been

removed, the margins of the bone cavity are cut away so that no overhanging edges remain and a smooth troughing of the bone results (Fig 44, *A*) This may be treated by allowing the soft parts to fill the cavity or a plastic muscle flap may be inverted and anchored by a few sutures (Fig 44, *B*) Primary suture of the overlying structures, or partial suture with drainage, is not recommended The former almost always fails and the latter is attended by infection and subsequent sinus formation Some form of open treatment is definitely preferable Many surgeons pack the wound

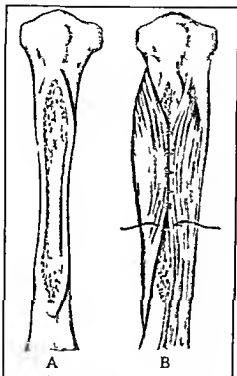


FIG. 44 — *A* Saucerization of shaft in chronic osteomyelitis *B* obliteration of cavity by muscle inversion

with iodoform gauze and renew the packing until healing obtains by granulation Bone repair results from the osteoblastic tissue lining the cavity and especially from periosteal osteogenesis Others favor secondary suture of the soft parts after the wound has been sterilized by the Carrel-Dakin technic When successful, the convalescent period is greatly shortened In certain cases of extensive diaphyseal involvement in a paired bone (radius, ulna, tibia or fibula) subperiosteal resection is advocated The periosteum is carefully elevated from the entire shaft and the diseased diaphysis



is removed care being taken to preserve all healthy involucral elements. A new shaft is regenerated by the cambial layer of the periosteum. In young children this forms rapidly and a substantial shaft is reproduced.

**Orr Technic**—The method advocated by Orr (1923) has gained much favor. Its underlying principles comprise adequate drainage and prolonged absolute immobilization. Only one operation is performed at any stage of the disease. The bone is exposed by a wide incision and all necrotic osseous tissue is meticulously removed. The cavity is then saucerized, thoroughly dried and packed with vaseline gauze. Dry dressings are applied and the extremity including the joints above and below is enclosed in a plaster cast in the neutral position. (Orr emphasizes the importance of absolute rest and advocates braces for months after healing has occurred.) The dressing within the cast is undisturbed for four to eight weeks unless severe pain or hyperpyrexia develops. The offensive odor is inconsequential. All antiseptics are interdicted, also frequent dressings as the latter may introduce secondary infection. The vaseline gauze is gradually extruded, often with small sequestra adherent to it. The immobilized joints regain function with usage.

**Recrudescences**—In many healed cases of chronic osteomyelitis recrudescences occur from time to time accompanied by acute pain and pyrexia. Trauma is a common provocative. Immediate surgery is meddling and often unnecessary as most cases subside under adequate immobilization and wet dressings.

**Sterilization of Wounds** Sterilization of osteomyelitic wounds is best accomplished by the Carrel Dakin technic. The solution is often irritating to the tissues of young children and should be diluted. Vaccines and bacteriophages appear to be definitely valuable in certain cases. The maggot treatment is not recommended. When employed a preliminary prophylactic dose of tetanus anti-toxin should be administered.

**Osteomyelitis of the Irregular Bones**—This condition is often complicated by joint infection and is best treated by ultraconservative waiting. Operative trauma may incite fulmination. In time the sequestered segments are either absorbed or extruded and healing follows.

In osteomyelitis of a long bone complicated by pyarthrosis attention should be directed to the joint rather than to the bone lesion. However when necrotic bone segments protrude into a joint they should be removed together with all traces of dead bone and granulation tissue. The joint is then maintained in a position best suited for usage as ankylosis generally follows.

**Osteomyelitis of the Skull**—Infection of the flat bones of the skull presents certain specific manifestations. It may result from trauma, sinus disease or be of hematogenous origin. Depending

upon the pathogenesis, the osteomyelitis may be localized or diffuse. In the former, generally a sequel of scalp wounds, the infected area is usually small and confined to the outer table. With sequestration, small spicules are extruded. The diffuse or spreading type is due to thrombophlebitis of the diploic veins. Thrombotic obstruction of these large radicles produces necrosis of both the inner and outer tables. Epidural and perierianial abscess often develop therefrom and at times subdural abscess or meningitis. With subsequent sequestration, large portions of the bone may be extruded. Osteoblastic activity is at a minimum or entirely lacking in such cases and the bone defect is permanent.

### EPIPHYSITIS

#### (OSTEOCHONDROSIS.)

Epiphysitis may develop in any ossification center during the period of active bone development. The varied etiologic factors comprise trauma, infection, lues, endocrine disturbances and avitaminosis. At times the causative factor is obscure.

Trauma may be followed by hemorrhage with resulting necrosis, fibrous tissue development and loss of the epiphyseal cartilage. Endocrine dysfunction of the pituitary, parathyroid or adrenal glands (possibly also of the thyroid) may cause disturbances of both growth and ossification. Infections are discussed under "Osteomyelitis," lues under "Syphilis of Bone," and vitamin deficiencies under "Rickets" and "Scurvy." Depending upon the pathogenesis, definite evidences of epiphysitis may be exhibited roentgenologically.

**Symptomatology.**—Although the symptoms vary with the type of pathology, they are chiefly those of pain, disability, local tenderness, swelling and deformity. Recovery generally follows rest, appropriate support and removal of the cause.

**Osteochondritis Deformans Juvenilis Coxæ (Legg's Disease, Perthes' Disease, Coxa Plana, Pseudocoalgia).**—The disease occurs in the second decade, chiefly in adolescent boys, and is characterized by changes in the epiphysis of the hip. Of unknown origin, it has been attributed to infection, tuberculosis, rickets and congenital defects of the femoral head and acetabulum.

**Pathology.**—The most distinctive features are flattening and fragmentation of the head of the femur. The flattened head may bulge beyond the acetabular margin and the joint cavity contain an excess of synovial fluid. In advanced cases there is progressive necrosis of the central portion of the epiphysis, followed by sequestration. The process may extend through the epiphyseal plate into the femoral neck and the acetabulum may also become irregular. The staphylococcus has been isolated frequently from the necrotic bone.

**Symptomatology** —The onset is insidious. Most often a lump or slight pain attracts attention. (The roentgen rays may exhibit a disproportional amount of pathology.) Examination in the early stages reveals limitation of abduction and internal rotation; later the thigh and gluteal muscles exhibit atrophy. Shortening may also develop from involvement of the epiphyseal cartilage and femoral neck.

The earliest roentgenologic finding is flattening of the femoral head. Later the epiphysis may become irregular, the neck thickened and the articular outline blurred. With regeneration normal density is restored in the neck and head but the latter remains permanently flattened.

**Diagnosis** —The paucity of symptoms in relation to the extensive pathology is often striking. Tuberculosis is associated with more pronounced symptoms: pain, muscle spasm, night cries and slight evening pyrexia, also greater destruction, rarefaction and atrophy are exhibited roentgenologically. Repeated negative tuberculin tests exclude tuberculosis.

**Treatment** —In early cases satisfactory bone regeneration is effected by rest in bed with ample sun or artificial ultra violet irradiation. This should be supplemented with a dietetic hygienic regimen: fresh air, high caloric diet, hematinics, fish liver oils or their concentrates and viosterol. Advanced cases require plaster spica immobilization of the leg in abduction and external rotation for several months. The treatment should be continued until adequate bone regeneration is demonstrable in serial roentgenograms. The ultimate outcome is good; at times there is slight residual limitation of abduction and internal rotation.

## SYPHILIS OF BONE

Syphilis of the osseous system in infants and children is almost always congenital or hereditary. In the former infection occurs from the luetic mother and in the latter from either parent or both.

The lesions are tertiary manifestations of gummatous formation and comprise osteochondritis, periostitis, osteitis, osteomyelitis and arthritis. The bones most commonly affected are the tibia, skull and phalanges and less frequently the sternum, metacarpal and tarsal bones.

**Osteochondritis** —The condition is often present in congenital lines being manifested by sensitive swellings in the epiphyseal regions at times accompanied by synovitis. The granulomatous infiltration of the epiphyseal cartilage and metaphyseal zone results in necrosis and in advanced cases epiphyseal separation often occurs. The latter may be confused with fracture. Regeneration follows appropriate treatment. In older children a chronic form of osteochon-

dritis occasionally develops which simulates tuberculosis (von Gies' joint). Sequestration and sinus formation may follow with resulting ankylosis. The clinical course is relatively painless and afebrile.

**Periostitis.**—Periosteal invasion by the spirochete occurs chiefly during the fifth and sixth years and most commonly involves the tibia. The proliferative inflammation produces a painful localized thickening over the shaft which frequently develops new bone or



FIG. 45.—Tuberc osteochondritis

hard nodules. The process may traverse the Haversian canals of the cortex and involve the cancellous tissue and marrow with resultant diffuse hyperostosis and curving of the tibia (*sabre tibia*). Conversely, the granulomatous tissue may primarily invade the marrow and spread to the cortex (*osteomyelitis*). Destructive involvement of the bony and cartilaginous framework of the nose results in *saddle nose*.

**Arthritis**—In early syphilis, nocturnal arthralgia may occur without structural or functional changes. Hydrarthrosis, characterized by simple serous effusion may also develop. The condition is often symmetrical and involves chiefly the knees, elbows and wrists. Bilateral involvement of the knees (Clutton's joint) may be associated with interstitial keratitis. In late syphilis, gummatous osteochondritis, osteoarthritis and osteomyelitis frequently occur concomitantly. The proliferative and destructive processes extend not only to the joint surface and articular cartilage but may also



FIG. 46—Syphilitic osteoperiostitis of the humerus causing bowing

involve the periarticular structures. The joint becomes swollen and tense, the overlying skin pale and shiny, but there is little pain or limitation of function. The process is definitely less destructive than tuberculosis and rarely results in ankylosis.

**Dactylitis**—Syphilitic dactylitis is often confused with tuberculosis. It is painless, often multiple, more proliferative than destructive, and exhibits little tendency to suppuration or sinus formation.

**Charcot's Joint**—Juvenile tabes' is rare. It is characterized by marked bony outgrowth, synovitis, and painless joint instability.

*Syphilitic Stigmata*—Certain stigmata are usually exhibited at four to six weeks the child may develop rhinitis snuffles pemphigous eruptions of the palms and soles fissures and mucous patches about the mouth and anus and enlargement of the liver and spleen. The first dentition is delayed and the second faulty. Hutchinson's teeth pegged teeth Fournier's teeth or the tubercle of Carabelli may be evidenced. Interstitial keratitis and eighth nerve deafness occur most commonly at puberty. Adenopathy of the epitrochlear and posterior cervical nodes occurs very frequently.

**Diagnosis** This may be made by identifying the treponema or by the complement fixation Wassermann and flocculation tests. The roentgenologic findings are those of bone destruction and proliferation. Luetic osteochondritis exhibits enlarged and irregular cartilage plates but not the saucer like appearance of rickets. A defect in the metaphysis is also commonly present (Fig 45). Bone atrophy is unusual and any change in shape is due to the laying down of new bone (Fig 46).

**Treatment** Specific therapy comprises the intramuscular administration of neoursphenamine (neosalvarsan) supplemented by bismuth or mercurial injections mercurial inunctions or biniodide of mercury orally. Surgery is seldom indicated except for extensive bone destruction or plastic repair of nasal and mouth lesions. Cases of non union following fracture which are uninfluenced by antiluetic treatment may require bone transplantation.

## CHAPTER VIII

### BENIGN BONE GROWTHS

BENIGN bone growths reflect the general features of bone development and in many respects are caricatures of normal osteogenesis.

The outer layer of the periosteum is composed of fixed fibroblastic cells and the inner of osteoblastic. Tumors derived from the former remain definitely fibroblastic, whereas those originating from the latter produce osteogenic growths. Other sites containing osteoblastic tissue from which osteogenic growths may arise are the inner surface of the cortex, the Haversian canals, the endosteum and its trabeculae.

Discussion of the growths will be in accordance with the nomenclature and classification of the American College of Surgeons.

#### 1 Benign osteogenic tumors

- (a) Exostosis
- (b) Osteoma
- (c) Chondroma
- (d) Fibroma

#### 2 Inflammatory conditions that may simulate bone tumors

Myositis ossificans

Osteoperiostitis

- (a) Traumatic
- (b) Syphilitic
- (c) Infectious

Ostitis fibrosa

#### 3 Benign giant-cell tumor

#### 4 Angioma (benign)

### EXOSTOSIS

Exostoses are irregular outgrowths of bone to which the term tumor is not strictly applicable.<sup>1</sup> The growths develop commonly from the periosteum (exostoses) and very rarely from the endosteum (enostoses). Arising in bones preformed in cartilage, they generally occur as multiple cartilage-capped tumefactions on the shafts of long bones near the epiphyses. A single exostosis is unusual. At times the growths present a symmetrical arrangement (epiphyseal aclasis).

<sup>1</sup> Deer antlers are the most remarkable exostoses occurring in the animal kingdom. These extensive growths of perfect bone structure are developed within three or four months and are shed and reproduced annually.

**Etiology** —The tumors appear to be associated with some growth disturbance which produces an abnormal proliferation of cartilage in the region of the epiphyseal plates. Developing as abortive growths from independent bone centers they become relatively farther removed from the epiphysis as the shaft lengthens. They have also been attributed to misplaced periosteal cartilage rests.

**Histology** —An exostosis represents all the phases which occur in the genesis of normal bone preformed in cartilage. Beneath the



FIG. 47 — Multiple exostoses

hyaline cap calcified cartilage rests upon an osseous base, and the central part containing marrow elements and fat may communicate with the medullary cavity. Growth and ossification of the tumefactions usually cease with full skeletal development. Although the tumors are essentially benign they are nevertheless subject to sarcomatous degeneration in certain instances.

**Symptomatology** Exostoses are probably present at birth but are seldom recognized before the fifth year. Between them and



puberty the small masses increase to various sizes and shapes and at times inhibit normal bone growth. Whereas the growths may occur in any of the bones preformed in cartilage the long bones of the extremities and the short long bones of the hands and feet are involved most commonly. Adventitious bursæ may cover the tumefactions and communicate with an adjacent joint. The subungual bony tumor occurring beneath the nail of the great toe is a solitary exostosis which develops from the inner aspect of the terminal phalanx.

**Diagnosis** — Multiple exostoses occurring in early life are readily diagnosed roentgenologically (fig 47). Solitary growths may at times be puzzling. A circumscribed encapsulated slow growing osseous tumor which arises from and is directly continuous with the cortex and which does not produce bone erosion is manifestly benign. At times an irregular calcification zone is demonstrable.

**Treatment** — Slow growing exostoses which do not produce pain dysfunction or disfigurement may be disregarded as their growth ceases with full skeletal development. In rare instances they regress and disappear spontaneously. Rapid growing tumors however connote malignancy and require prompt extirpation. The growth should be completely removed and the base cauterized to prevent recurrence. Amputation may be elected for large phalangeal exostoses.

## OSTEOMA

Osteomas are tumors composed of bone and arising from bone. Although the appellation should be restricted to true neoplasms histologic study at times fails to distinguish simple hyperplastic growths from true osteomas. The latter are formed either by direct proliferation of osteoblasts or through an intermediate cartilage stage. False osteomas resulting from metabolic disturbances or associated with inflammatory processes develop as flat flakes or spur formations and are designated *osteophytes*. Sharp prominences due to periostitis or osteomyelitis are termed *spurs*.

True osteomas occurring as independent progressive bone tumors unassociated with inflammatory conditions are uncommon. *Con genital cases have been recorded. Due to very slow growth rate,* the occasional osteoma developing in childhood often escapes attention until adult life. The genus contains two species compact and cancellous osteomas.

**Compact Osteomas** — These hard tumors arise chiefly from bones developed in membrane. Of the flat bones of the skull the frontal and parietal are most commonly involved. The growths are usually sessile of eburnated consistency and grow very slowly (Fig 48). Histologically the exceedingly dense compact bone exhibits a variable degree of osteoblastic activity. The tumors also occur in the

vertebrae frontal and maxillary sinuses roof of the orbit external auditory meatus and mandible (Fig 49) Those of the frontal



FIG 48 —Osteoma of the skull fitted covered at four years of age



FIG 49 Osteoma of the frontal sinus

sinus and of the orbit occasionally attain a remarkable size Many mandibular tumors diagnosed osteomas prove to be odontomes

**Cancellous Osteomas (Spongiosum)** — Cancellous osteomas mimic the structure of cancellous tissue. Occurring more frequently than the compact type, their production is limited to the period of active bone growth. They appear to be the product of some irregularity or defect of bone development, possibly a minor degree of dyschondroplasia. Occasionally there is a familial history. The growths occur as rounded or mushroom shaped tumors in the vicinity of the epiphyses of long bones (Fig 50). At times they are multiple. The dome of the tumor is generally capped with cartilage and growth continues until the cartilage ossifies. Occasionally the tumors are grooved for the passage of tendons.

**Symptomatology** — Osteomas are painless tumors of very slow growth and often fail to attract attention before attaining large size. They may be discovered accidentally or dysfunction or reflex pain may be the first symptom. The latter is especially common in growths of the frontal or maxillary sinuses (Fig 49). The tumors at times become detached spontaneously.

**Diagnosis** True osteomas are characterized by very slow growth, positive roentgenologic findings and the absence of inflammation. Histologically they exhibit evidence of definite osteoblastic tissue proliferation. False osteomas are associated with inflammatory lesions, are limited in size and reveal slight or no osteoblastic activity.

**Treatment** — Being definitely benign tumors of exceedingly slow growth, osteomas require no treatment unless they produce pain, deformity or dysfunction. Eburnated growths require good instruments for removal.



FIG 50 — Cancellous osteoma of tibia



FIG 51 —Multiple chondromas in the same patient

genograms revealed a second chondroma in the right os magnum (Fig 53)

**Malignancy Potential**—Although essentially benign, chondromas are related clinically to sarcoma. They are notoriously persistent



FIG 52—Before and after operation

and recurrences following incomplete surgical removal may eventuate in metastases. The roentgen ray of B S, aged seventeen years, is illustrative (Fig 54). The boy complained of pain in the head

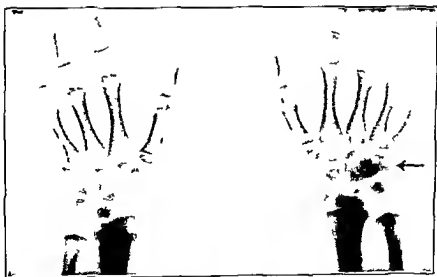


FIG 53—Chondroma of os magnum in the same patient

of the left tibia for six months. Examination revealed a non tender ovoid swelling of the upper third of the tibia which exhibited an egg-shell crackle. At one point a sinus discharged sticky purulent

material. Following roentgenographic study the presumptive diagnosis was chondroma. At operation the growth involved the upper third of the diaphysis being limited superiorly by the epiphysis the posterior surface of the shaft was also eroded. Following enucleation the walls of the cavity were cauterized and the wound was packed with iodoform gauze. Pathologic report hyaline chondroma. Infection followed which subsequently involved the knee-joint. Amputation was performed four months later. The growth at this time revealed osteogenic sarcoma. Death resulted from pulmonary metastases.

**Diagnosis.** The history is irrelevant the chondroma may be discovered accidentally or attention be attracted to it by pressure symptoms dysfunction or rarely through spontaneous fracture. Multiple chondromas and the solitary encapsulated lobulated type which develops in the epiphyseal ends of the diaphyses of long bones are readily diagnosed roentgenologically. Atypical cases simulating osteitis fibrosa cystica bone cysts or giant cell tumors may require punch biopsy or exploration for diagnosis. (The cartilage which occasionally occurs in mixed tumors of the salivary and lachrymal glands the breast and testis does not pertain to the genus chondroma.)

**Treatment.** — Asymptomatic slow-growing tumors require no treatment. Acceleration of growth rate connotes malignancy and for that reason rapid growing chondromas demand prompt removal. The tumor and its capsule should be completely extirpated and the cavity cauterized with carbolic acid or the actual cautery. Except in pedunculated growths it is advisable to remove too much rather than too little of the adjacent bone as recurrences are common after incomplete excision. They are extremely dangerous.

**Ecchondroses.** — The tumors are small local outgrowths from pre-existing cartilage. They occur uncommonly and develop chiefly



FIG. 54. Hyaline chondroma of the femur with erosion of bone. Tumor is encapsulated in sarcoma.

at the costo-sternal junctions in association with rickets along the edges of articular cartilages and on the laryngeal and nasal triangular cartilages. Trauma and inflammation are common etiologic factors.



FIG. 33 — Multiple osteochondroma of the radius causing retardation of growth

The growths occur as small smooth irregular tumefactions and are either discovered accidentally or attract attention through dysfunction or pain. Their histologic pattern is the same as the cartilage from which they develop. Asymptomatic osteochondromas may be disregarded.

**Synovial Condromas** The villi of large joints which become hypertrophied through chronic infective or metabolic disturbances are at times subject to hyaline and calcification. Although pure hyaline villi may result therefrom an admixture of calcific granules generally occurs. The resulting nodules may become detached and produce joint mice. The pathology is essentially one of adult



FIG. 50 —Multiple osteochondroma producing pressure atrophy of the fibula



FIG. 51 —Osteochondroma of the femur first discovered at twelve years of age



life and arthrotomy for the removal of loose synovial chondromas is seldom required in childhood

**Osteochondromas**—The tumors are composed of both osseous and cartilaginous elements. They may develop primarily as osteochondromas or result from osteogenesis in a preexisting cartilage tumor. The usual type is composed of poorly calcified hyaline cartilage surrounded by an osseous or fibrous tissue capsule.



FIG 58 Solitary osteochondroma

At times congenital, the growths generally develop during childhood and involve chiefly the shafts and joint ends of long bones. When multiple tumefactions occur, some may be regressive while others are enlarging. The tumor growth usually ceases with full skeletal development.

**Multiple Tumors**—Extensive multiple osteochondromas are probable related to chondroplasia and are occasionally familial. The tumors frequently inhibit bone growth and extreme stunting therefrom may result in dwarfism. Retardation of radius growth may produce an ugly deformity (Fig 55), and an arrest of fibula growth, *pes valgus* (Fig 56).

**Histology**—The tumors exhibit a persistence and overgrowth of poorly calcified cartilage in which the cells are disorderly arranged in

variable size and form. At times the epiphyseal plates are obliterated and multiple osteophytes may develop from prolonged ossification. Although essentially benign osteochondromas may rarely undergo sarcomatous degeneration.

**Diagnosis**—Multiple and typical solitary tumors are readily diagnosed roentgenologically. They are definitely encapsulated and often exhibit osteoporosis and a polycystic appearance with more or less pronounced interlobular septa (Fig 57). Although the compact bone of the shaft ends may be deficient there is no evidence of bone infiltration or destruction (Fig 58).

**Treatment** —Slow-growing tumors which do not produce dysfunction pressure symptoms or arrest of bone growth should be kept under observation. Occasionally irradiation will produce regression and partial absorption. Rapidity of growth however should always arouse the suspicion of malignancy. In such instances complete extirpation of the tumor with a wide zone of bone about the base should be performed promptly. Recurrences are dangerous and may be followed by malignant metastases.

### FIBROMA

Periosteal fibromas of the long bones are uncommon and are usually associated with trauma. Pure medullary types are exceedingly rare. *Fibrous epulis* of the maxilla generally projects into the mouth although in rare instances the tumor may develop in the interior of the bone from developmental dentitional disturbances (Refer to Epulis). Fibromas are frequently subject to hyalinosis or calcification and at times to osteoblastic changes.

### MYOSITIS OSSIFICANS TRAUMATICA

The condition is a localized deposition of bone in the muscles or about their insertions. Although trauma is the chief etiologic factor there is probably also an associated diathesis or predisposition to osseous formation. The process occurs most often in the brachialis anticus following posterior dislocation of the elbow and in the adductor muscles of the thigh (rider's bone).

**Pathology** —The proliferation of both cartilage and bone appears to be stimulated by the extravasated blood in periosteal tears and muscle trauma. Chondroblastic activity develops in the organization of the clot and the resulting cartilage becomes transformed into bone (ossifying hematoma). Histologic examination may exhibit new cartilage and bone in various parts of the organized clot. The period of osteogenesis corresponds to that occurring in callus, the osteoid tissue developing in about eighteen days and the calcification requiring an additional two weeks.

**Symptomatology** —The swelling following severe trauma usually subsides within a fortnight and persistence thereafter may indicate a beginning ossifying myositis. As the process develops the tumefaction becomes progressively firmer, at times increases in size and finally attains the hardness of bone. After four or five weeks, horizontal deposits of bone parallel to the shaft axis are demonstrable radiologically. (Figs 59 and 60)

The condition is painless unless nerves are involved. Progressive dysfunction may occur when the ossification develops in juxtaposition to a joint. The process may remain stationary or regress and subsequently disappear.

**Diagnosis** —Although a subperiosteal ossifying hematoma may mimic myositis ossificans clinically, the roentgenogram of the former exhibits the broad base of the mass in contact with the shaft. Periosteal osteogenic sarcoma may follow trauma and in the early stages simulate myositis ossificans. The perpendicular striation in the former and horizontal lamellations in the latter are important differentials. As the process advances, sarcoma exhibits progressive and destructive growth whereas myositis ossificans remains stationary or becomes regressive.

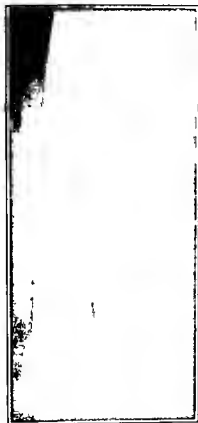


FIG. 59 —Early myositis ossificans traumatica



FIG. 60 —Late myositis ossificans traumatica

**Treatment** —Rest and cold compresses are indicated in the early stage and at a later period, diathermy and massage. After ossification has occurred, the mass should be observed for several months as complete resolution may follow. If regression does not occur and symptoms of pain or dysfunction develop the tumor including a layer of surrounding tissue should be excised.

## MYOSITIS OSSIFICANS PROGRESSIVA

In this rare condition of unknown etiology multiple nodules of bone develop in the muscles and subcutaneous tissues (Fig 61)



Fig 61 — Myositis ossificans progressiva.

The pathology has been attributed to a defective development of the terminal capillaries which results in multiple hemorrhages and subsequent ossification.

As the disease advances the osseous masses gradually increase in size and number. The process is essentially painless unless nerve endings are irritated. Defective stature, infantilism and microdactylia are frequently associated with the condition.

**Treatment** Painful nodules may require excision. Diets low in calcium and phosphorus, iodides and glandular therapy have all been disappointing. Therapeutic irradiation is of dubious value.

### OSTEITIS FIBROSA

Osteitis fibrosa occurs so predominantly in children that Long termed the condition *osteodystrophia juvenilis*. It is characterized



Fig. 62.—Osteitis fibrosa cystica of the humerus. Cortical fracture following trauma.

by defective calcification and the development of fibrous tissue and cysts in one or many bones of the limbs, jaws, trunk or skull. The process occurs chiefly in the shafts of long bones, especially the upper thirds of the humerus, femur or fibula, and pursues a chronic course. Its incidence is greater in males. (In elderly patients the disease is apparently related to osteomalacia and possibly to Paget's osteitis deformans.)

**Etiology**—The cause or causes remain undetermined. In some instances there is a history of trauma and in others hyperparathyroidism appears to be a factor.

Maudl (1926) effected a cure in osteitis fibrosa cystica by removing a parathyroid tumor. Several cases have subsequently been reported by Barr, Bulger, Churchill and others in which the hyperparathyroidism was associated with a parathyroid adenoma or with diffuse hypertrophy and hyperplasia of the glands.

**Pathology**—Bone tissue is replaced by cellular fibrous tissue which invades the Haversian canals and bone-marrow. The shaft may become expanded thereby and the cortex thinned, the periosteum however remains intact. Occasionally new bone is formed. The process rarely involves the epiphyses.

The cellular tissue may soften and produce multiple cysts, lined by fibrous tissue and containing clear fluid (osteitis fibrosa cystica). Not infrequently a few giant-cell areas line the cyst walls and in some instances with or without cyst formation, a giant-cell tumor develops. The cells are of the epulis type and are probably of inflammatory character (Refer to Giant-cell Tumors). Some observers consider osteitis fibrosa cystica and giant-cell tumors to be the residuum respectively of regressive and aggressive changes.

**Symptomatology**—Intermittent or constant mild aching pain is usually the first symptom. Tenderness and swelling are variable. In some instances a fracture occurring spontaneously or following slight trauma, attracts attention to the condition (Fig 62). In the generalized type similar changes occur in many bones and the process may be more extensive (Fig 63).

**Diagnosis**—Osteitis fibrosa cystica can usually be readily diagnosed roentgenologically. The small medullary cyst areas are separated by fine trabeculae and the cortex frequently appears thin and bulging beneath an intact periosteum. Giant cell tumors, which rarely develop before the age of fifteen years, occur almost always in the epiphyseal ends of long bones and exhibit denser



FIG 63—Extensive osteitis fibrosa cystica improved by parathyroidectomy (Courtesy of Dr. W. H. Irish.)

trabeculation. Medullary gumma may be confusing. In addition to a positive Wassermann reaction, luetic stigmata may also be present. A sharply outlined intramedullary rarified area, not accompanied by fever, is highly presumptive evidence of osteitis fibrosa.

**Treatment.**—In the absence of pain, progressive growth, increasing deformity, or danger of fracture in a weight-bearing bone operative interference is contraindicated. Roentgen irradiation often produces regression and in rare instances spontaneous resolution occurs. Pathologic fractures generally unite although calcification of the callus is delayed.

Operative measures comprise the removal of the cysts and curettage and cauterization of the cavity with 95 per cent phenol neutralized with alcohol. When possible the dead space should be obliterated by collapsing the walls. In cases of extreme cortical destruction subperiosteal resection may become necessary. A bone graft implant is then required for stabilization. Although osteogenesis is slow, the surgical results are usually excellent and recur-

turbed, the process pursues a slow but progressive course. Extensive tumors occasionally prove lethal from hemorrhage or infection. The genus is definitely benign and does not metastasize.

**Varieties of Giant Cells**—Three types of giant cells occur in pathologic lesions. (1) In rapidly growing malignancies giant cells may be the result of karyorrhexis, the cytoplasm being unable to keep pace with the rapidity of nuclear division. The multiple nuclei are irregular in size, shape and distribution. (2) The so-called "foreign body" type of giant cell, occurring in tuberculosis and gumma, exhibits multiple nuclei which are either bipolar or eccentric in distribution. (3) The giant-cell tumor type in which the multiple nuclei are of similar form and size, completely separated from each other and occupying a central position in the cytoplasm.

The term "giant-cell tumor" is restricted to lesions in which the giant cells are predominantly those of Type 3. Clinically such skeletal lesions occur in (a) osteitis fibrosa, (b) epulis, and (c) giant cell sarcoma. The giant-cell growth occurring in osteitis fibrosa is generally considered to be of inflammatory character. Epulis tumefactions arising in the gums or alveolar paradontium are definitely benign tumors and the term "giant cell of epulis type" is often employed to emphasize the benignity of the genus. Whether giant-cell sarcoma is definitely neoplastic or the response product of inflammation remains controversial.

**Etiology**—There is often an antecedent history of trauma and many consider the process a sequel of hemorrhagic osteomyelitis. All giant-cell tumors, however, cannot be attributed to an initial hemorrhage. Some apparently result from osteitis fibrosa cystica and others from the absorption of islands of cartilage which persist in the epiphyses in rickets and other bone disease. At times there is no demonstrable etiologic factor and the growths appear to occupy a mid-position between that of inflammation and tumor.

**Incidence**—From Fig. 64 it is evident that giant-cell tumors are uncommon before puberty and usually develop between the ages of sixteen and twenty five years. They occur chiefly where bone growth has its greatest momentum—in the lower end of the femur, upper end of the tibia, upper end of the humerus and lower end of the radius, also in the vertebrae os innominatum and inferior maxilla. They seldom originate in the shafts of long bones or in those preformed in membrane. Any bone may be involved, however, and in rare instances the growths are multiple.

**Pathology**—Typical tumors developing in the epiphyseal ends of long bones produce an expansile excavation of the cancellous tissue and a thinning of the shaft (Figs. 65 and 66). This results from concomitant destructive and productive processes. While the growth tissue is destroying the medullary elements and cortex, the periosteum lays down new bone. The tumor, destructive but not



infiltrative, remains encapsulated within the periosteum and its new-formed bone shell, sharply demarcated from the normal shaft. Cartilage acts as a definite barrier and a growth occurring before the epiphyseal plate ossifies is generally limited thereby. Although the epiphysis may be crowded by tumor tissue, the articular cartilage remains intact. The neighboring joint is rarely involved but at times may contain a clear exudate.

**Gross Pathology**—This depends upon the tumor's phase. In active growing aggressive types the tumor mass is composed of vascular current jelly like granulation tissue, rather firm in con-

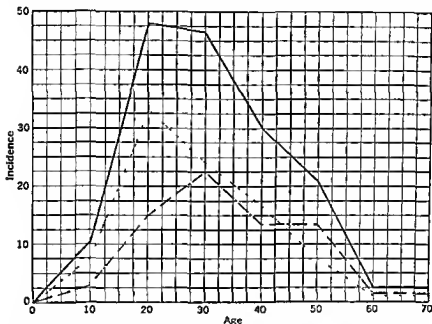


FIG. 64—Curve illustrating the incidence of giant cell tumor in relation to age. dotted line females broken line males heavy line both sexes combined. (After Kolodny, courtesy of American College of Surgeons.)

sistency and mottled with white areas. Upon division of the capsule the tumor tissue tends to extrude and oozing may be pronounced. The fibrous tissue stroma forming the polycystic walls is easily broken up except when calcified. With regression or following radiation therapy the tumor mass becomes denser through attendant fibrosis and its central portion frequently contains cysts. In advanced growths with shaft involvement and thinning of the cortex pathologic fracture may occur.

**Histology**—The tumor consists of stroma and giant cells. The former is composed of numerous capillaries and blood spaces supported by a loosely woven network of spindle round or polygonal cells. Pleomorphism does not occur. The giant cells, few or many,



FIG 65

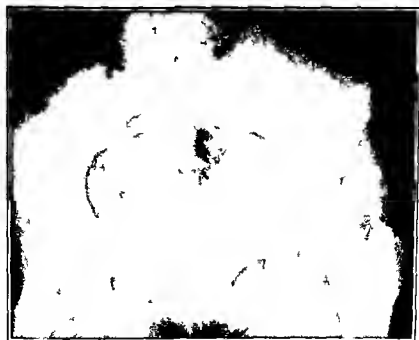


FIG 66

FIGS 65 and 66 —Giant-cell tumor of femur before and after bone pegging  
(Courtesy of W H Irish)

are loosely embedded in the stroma. These large opaque acidophile cells contain multiple small oval nuclei of equal size distributed throughout the central portion of the cytoplasm. The cells often appear like knots at the junction of endothelial strands and are thought to be of endothelial origin. Although the tumor capsule may exhibit osteoblastic activity atypical bone formation in the tumor substance connotes malignancy. It is remarkable that such vascular structures do not metastasize even after treatment by curettage.

**Symptomatology**—Constant pain of a dull boring character is usually the first symptom followed by progressive disability. Later a swelling may develop. In other instances due to slow growth rate and lack of intraperiosteal tension swelling may first attract attention. Tenderness is less constant than in osteogenic sarcoma. Advanced growths in weight bearing bones occasionally cause pathologic fracture either spontaneously or from slight trauma.

**Diagnosis**—The diagnosis can usually be made by roentgenologic study. The salient characteristics are those of a slow growing circumscribed multiventricular tumor in the epiphyseal end of a long bone sharply demarcated from normal bone and producing a widening of the shaft with preservation of the intact periosteum and an absence of periosteal lifting. The interlobular septa producing the soap bubble appearance are generally more pronounced than in osteochondroma. Giant-cell tumors seldom develop before puberty whereas osteitis fibrosa occurs most commonly in childhood. Osteogenic sarcoma is of more rapid growth and exhibits bone destruction and infiltration. (Refer to Sarcoma.) Roentgen therapy definitely inhibits growth and produces regression in giant-cell tumors. Obscure cases may require punch biopsy or exploration for diagnosis.

**Surgical Treatment**—Although Nelaton recognized the benign character of giant-cell tumors many unnecessary amputations have been performed in the mistaken belief that the growths were lethal sarcomatous processes. Bloodgood's epochal report (1910) of a large series of cures following curettage of the growth further emphasized the benign character of these tumors. Thorough extirpation of the tumor mass followed by cauterization of the cavity by either the actual cautery or 90 per cent phenol neutralized with alcohol usually eventuates in cure.

**Radiation**—Operative treatment however is not always an ideal procedure for it may be followed at times by infection or recurrence of the growth. Moreover deformities and variable degrees of dysfunction are frequent sequelae. The more recent introduction of radiation therapy offers a positive and valuable advance. Tumor growth even in advanced stages can be definitely controlled thereby and response by low absorption usually follows with preservation of function.

### ANGIOMA (BENIGN)

Angiomas of bone are rare. The abnormal overgrowth of blood-vessels generally originates in the medullary cavity rather than in the Haversian canals or beneath the periosteum. Histologically, a variable amount of stroma supports the mass of thin-walled blood-vessels. Although many cavernous angiomas arising from either the periosteum or bone-marrow are benign, extensive cavernous growths of the long bones, especially of the femur and humerus, are usually considered sarcomatous.

**Symptomatology**—Angiomas may be asymptomatic until they produce periosteal tension with resulting pain. In rare instances pathologic fracture may occur from erosion of the shaft. Preoperative diagnosis is often impossible.

**Treatment**—Some angiomas are radio-sensitive and for that reason preliminary therapeutic irradiation should always be tried. When regression does not obtain, the usual operative procedure comprises ligation of the main nutrient vessels, followed by excision or curettage of the growth and cauterization of the cavity. Extensive cavernous angiomas may necessitate amputation.

### BONE CYSTS

Primary bone cysts are usually solitary lesions and occur chiefly in the medullary cavities of long bones. There is often a history of trauma and the pathology may be the residuum of hemorrhage. This is further emphasized by the fact that the cysts usually develop during childhood when the marrow is highly vascular. Secondary bone cysts may result from regressive changes in chondromas, myxomas, giant-cell tumors, and especially in osteitis fibrosa. (Refer to Osteitis Fibrosa.)

**Pathology**.—Bone cysts are generally definitely encapsulated and their firm fibrous lining can often be peeled readily from the bone. They contain straw-colored fluid, actual blood connotes malignancy. The cyst walls may exhibit giant cells of the epulis type and contain calcific deposits. A zone of osteitis fibrosa frequently surrounds the lesion.

**Symptomatology**.—Cysts generally develop before adolescence and seldom after the age of twenty years. They occur most commonly in the medullary cavities of the long bones, phalanges and skull. Roentgenographic findings are usually quite characteristic (Fig. 67). Due to slow growth rate, the element of pain is seldom pronounced. In many instances the condition is unnoticed and a pathologic fracture may be the first symptom.

**Diagnosis**—Brodie's abscess in its early stages may be confused with a bone cyst. The former is usually more irregular in outline

and exhibits a surrounding reactive zone. Giant-cell tumors seldom occur before puberty, are lobulated and develop in the epiphyses. Chondromas are generally lobulated and multiple. Myxomas are more confusing. Exploration may be necessary at times for diagnosis.

**Treatment**—The cysts occasionally resolve spontaneously and operative interference is only indicated when there is pain, danger of spontaneous fracture or in cases of doubtful diagnosis. Pathologic fractures seldom require open reduction. They generally heal spontaneously but calcification of the callus is often tardy.



FIG. 67—Bone cyst of frontal bone

Operative measures comprise the removal of the cyst wall followed by curettage and cauterization of the cavity with 95 per cent phenol neutralized with alcohol. The cavity may be packed with iodoform gauze or obliterated by collapsing the surrounding osseous tissue. The bleeding is often profuse in extensive conditions and hemostasis should be secured by a tourniquet. Following the removal of large cysts in weight bearing bones a bone graft implant may be necessary for stabilization.

## ECHINOCOCCUS CYSTS OF BONE

### (HYDATID CYST)

Multiple echinococcus cysts of bone occur chiefly in the articular ends of the long bone, bodies of the vertebrae and the pelvic girdle. As the cysts enlarge the bone becomes expanded and the cortex finally perforates; secondary necrosis and suppuration follow.

**Symptomatology.**—The condition is afebrile in the absence of infection and the usual symptoms are deep-seated osteoscopic pain and swelling; upon pressure a "ping pong crackle" may at times be elicited. With secondary infection spontaneous fracture may occur. The cysts are readily demonstrable roentgenologically and are often associated with other cysts, especially in the liver. The blood exhibits eosinophilia.

**Treatment.**—In some instances, death of the scolices and absorption of the cysts have followed the intravenous administration of neosalvarsan. Operative measures comprise the removal of the cyst and all daughter cysts. If this is impossible the cyst should be first distended with tincture of iodine, 1 per cent formalin or ether for ten minutes, in order to kill the scolices. The germinal cyst wall is then removed and the wound marsupialized.

## CHAPTER XIV

### SARCOMA OF BONE AND BONE MARROW

DESTRUCTIVE tumors of bone and bone-marrow constitute one of the most important and complex problems of oncology. Prior to the Registry of Bone Sarcoma by the American College of Surgeons (Codman 1921) great confusion arose from the numerous and varied classifications of artificially segregated types and varieties. Nomenclature was predicated in large part upon the predominating cell type and pathologic reports of osteosarcoma osteochondrosarcoma myxochondrosarcoma etc were common. Specimens of a single tumor referred to different pathologists often resulted in a variety of histopathologic diagnoses. By adherence to the appended classification adopted by the Registry in 1923 comparable data may be accumulated for accurate evaluation.

#### 1 Osteogenic sarcoma

##### A Anatomic types

- (a) Medullary and subperiosteal
- (b) Periosteal
- (c) Sclerosing
- (d) Fungoid

##### B Undifferentiated sarcoma

#### 2 Periosteal fibrosarcoma

#### 3 Malignant angioma (angiosarcoma)

#### 4 Ewing's sarcoma

#### 5 Myeloma

#### 6 Metastatic tumors primary in tissue other than bone

#### 7 Benign giant-cell tumors

Osteogenic sarcoma is a tumor derived from the progenitors of cells which when fully developed are termed osteoblasts. The tumor tissue may thus exhibit various stages of osteoblasts from simple spindle cells to mucoid cartilage and true bone cells. A full appreciation of this concept is of the utmost importance. Bone cells are not necessarily produced yet the tumor cells possess the inherent potential to produce them. In slow growing tumors cell differentiation may ultimately attain bone-cell production whereas in rapid growths inhibition intermediate in differentiation may result in mucoid or cartilage cells. In rare instances only primary spindle cells occur in disordered arrangement.

Undifferentiated rapidly multiplying cells produce a lytic action

which destroys the involved bone. Thus in some parts of the tumor an area exhibiting undifferentiated spindle cells will cause bone absorption while in other parts where considerable differentiation has occurred cartilage or bone cells may be developing. Due to different stages of cell differentiation the majority of osteogenic sarcomas exhibit a conglomeration of various tissues of osteoblastic origin.

The first two subdivisions of osteogenic sarcoma (a) medullary and subperiosteal and (b) periosteal appear to depend upon an anatomic limitation by the periosteum. It is questionable however whether periosteal osteogenic sarcoma ever occurs without subperiosteal involvement. Moreover its microscopic appearance is the same as that of subperiosteal or medullary sarcoma and there is no evidence to indicate any difference in its growth progress.

Periosteal sarcoma is a term often employed because of the roentgenologic findings of lifting or wedge like lifting of the periosteum. This reactive response of the periosteum is not pathognomonic of malignancy. Its presence however definitely indicates subperiosteal invasion. In an or needle-like osteophytes perpendicular to the shaft may or may not occur. These are also demonstrable at times in low grade pyogenic and tuberculous osteomyelitis.

Sclerosing osteogenic sarcoma exhibits dense tissue with only an occasional tissue cell. However the cellularity and pleomorphism occurring elsewhere in the tumor connote the same grave prognosis as that of average osteogenic tissue. Early sclerotic tumors may later become osteolytic. Telangiectatic is a relative feature indicating great vascularity of the tumor.

Periosteal fibrosarcomas are definitely devoid of osteogenic tissue and do not invade the cortex. In the Registry cases there is no evidence to indicate an origin from the outer fixed fibroblastic tissue of the periosteum. They apparently develop from the periosteal fascicular layer and extraperiosteal would be a more descriptive term. Occurring most often at points of tendon insertions the tumors have a definite histologic pattern, slower growth rate and better prognosis than osteogenic sarcoma. (Refer to Extraperiosteal Sarcoma.)

Malignant angiomas are extremely rare if truly existent. Telangiectatic osteogenic sarcoma appears sufficiently inclusive for all highly vascular osseous sarcomas.

For clinical purposes primary bone malignancies fall into the following simple classification:

- 1 Osteogenic sarcoma
- 2 Ewing's sarcoma
- 3 Myeloma
- 4 Unclassified—including extraperiosteal fibrosarcoma and angio-endothelioma



## OSTEOGENIC SARCOMA

**Incidence**—Osteogenic sarcoma occurs twice as frequently as giant-cell tumor and affects approximately 1 in every 100 000 persons. It is primarily a disease of the young. The age and sex incidence based upon the Registry cases is illustrated in the following graph (Fig 68)

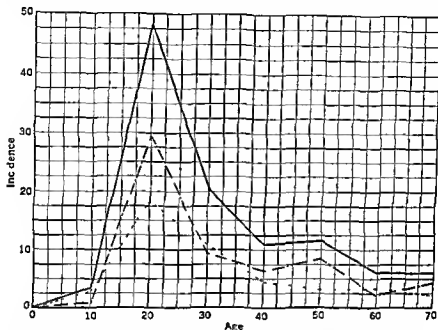


FIG 68 Chart illustrating the incidence of osteogenic sarcoma in relation to age. Dotted line females broken line males heavy line both sexes combined (After Kohn, courtesy of the American College of Surgeons)

The disease is accordingly rare in the first decade and reaches its maximum incidence at twenty years. No authentic case of congenital osteogenic sarcoma has been reported. Energetic skeletal development appears to be an etiologic factor.

**Situation**—The tumors develop most frequently in the metaphyses of long pipe bones. Approximately 70 per cent occur in the lower extremity and 10 per cent in the upper. Moreover there is a predilection for certain bones and over one-half the cases occur in the lower portion of the femur. Other femoral sites are the shaft, greater trochanter and neck. (Occurring in the shaft osteogenic sarcoma is frequently mistaken for Ewing's tumor.)

The upper third of the tibia, especially its inner aspect, is the next most common site (20 per cent of all cases). Growths of the shaft and lower end are uncommon. The lower femoral and upper tibial epiphyses are the last to ossify (twenty-one to twenty-two years).

Since over 70 per cent of sarcomas appear in these sites, it appears that prolonged growth rate may be a contributory factor.

Approximately 9 per cent of osteogenic sarcomas occur in the upper third of the humerus, a growth below the deltoid tubercle is very unusual. The glenoid region is a favorite site in scapular involvement. About 5 per cent are found in the pubic bones commonly about the iliac crest. Sarcoma of the fibula and ulna most

#### PRIMARY REGISTERED BONE TUMORS

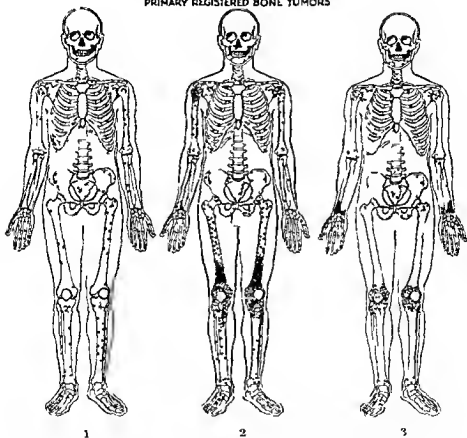


FIG. 69—1 Ewing's sarcoma. 2 osteogenic sarcoma. 3 benign giant-cell tumor.  
(Courtesy of American College of Surgeons.)

always involves the upper third of the shaft and that of the radius, the lower third. Although the metacarpal and metatarsal bones are subject to the disease the phalanges appear immune. Growths of the skull occur chiefly in childhood. The vertebrae and patella are very rarely involved, the ribs but seldom. (Fig. 69.)

Osteogenic sarcoma is essentially a solitary growth whereas Ewing's sarcoma is often multiple and meloma is generally so. At times multiple sarcomas becomes superimposed upon multiple

chondromas Loss of growth restraint may be a factor in such cases

**Pain**—This is commonly the first symptom and may precede the tumefaction by weeks or months *Persistent bone pain in a young subject should always excite suspicion of osteogenic sarcoma* The pain is apparently due to periosteal tension and often subsides after the growth perforates the periosteum It is usually a constant severe boring type and worse at night in some cases there is only an aching or tired feeling The degree of pain however is no index of growth virulence Intermittent symptoms suggest Ewing's sarcoma

**Trauma** In a minority of instances there appears to be a definite relationship between trauma and the development of osteogenic sarcoma In contradistinction to carcinoma single severe injuries are more provocative than repeated mild ones However the danger of trauma is extremely remote as is evidenced by the frequency of fracture and the relative rarity of sarcoma The average interval between a causative trauma and the development of pain or tumor is one to three months Approximately one-third of the cases present such history

The patient's general condition is seldom affected by the growth until general dissemination occurs Grave secondary anemia then follows and the patient develops a characteristic chalky appearance Whereas recurrent fever occasionally occurs in the early focal stage constant pyrexia follows metastatic diffusion The blood may exhibit a lymphocytosis with myelocytosis of 3 to 5 per cent

At times the size shape and consistency of the tumefaction is determinable by careful palpation and in certain cellular growths definite shrinkage follows irradiation After the tumor perforates the periosteum the growth rate often increases rapidly Egg-shell or ping pong crackle is an uncommon finding and bruit and pulsation are rare The overlying skin frequently becomes stretched and develops dilated veins In contradistinction to carcinoma the derma does not become adherent to the growth and ulceration rarely occurs There may be increased local heat

Pathologic fractures are uncommon as pain generally restricts the use of weight bearing bones Following such accidents however spontaneous healing may occur Cartilage acts as a definite barrier to sarcomatous invasion and the joints are seldom involved In advanced tumors involvement may occur from pericapsular extension or from a fracture into the joint

**Metastases**—Metastatic dissemination is a constant manifestation of osteogenic sarcoma Although this almost always occurs through the blood stream the lymph system is nevertheless capable of transporting tumor cells and in rare instances neighboring lymph nodes become involved Primary metastases develop most com-

monly in the lungs. Preceding the development of cough or signs of bronchitis multiple small diffuse shadows may often be exhibited roentgenologically. At times these disappear under irradiation. Later the metastases may attain large size. Secondary dissemination occurs commonly in parenchymatous organs but the osseous system is rarely involved. Metastases generally develop within thirty months although they may be delayed for several years.

**Diagnosis**—The early diagnosis of osteogenic sarcoma is at times extremely difficult. In some instances the history and clinical findings suffice while in others roentgenograms may be even more decisive than the microscopic pattern. Not infrequently a combined study of the clinical, roentgenologic and pathologic findings is required.

*Roentgenography is the most valuable preoperative diagnostic aid.* Although there are no findings pathognomonic of sarcoma certain roentgenologic evidences are highly presumptive. Due to periosteal and cortical resistance most osteogenic sarcomas develop as spindle form tumors. One of the most important signs in differentiation from a benign tumor is the absence of a definite limited outline.

Bone reaction and the degree of cell differentiation in the tumor influence the character of the roentgenologic evidence. Osteoblastic reaction with ossification may exhibit radiating bony spicules and this sun ray or fan like arrangement of newly formed bone is highly suggestive of sarcoma. The striations however occur in less than



FIG 70 Osteogenic sarcoma of the tibia exhibiting lipping

20 per cent of tumors and are occasionally present in chronic inflammations. Less frequently new layers of osseous tissue develop parallel to the shaft and simulate the longitudinal structures occurring in periostitis. At times the tumor appears spotty due to areas of increased osseous density.

Lipping or wedge like elevation of the periosteum is a common finding. This also occurs at times in pyogenic and tuberculous osteomyelitis. In osteolytic tumors the bone shadow becomes spotty and ultimately may be partially or totally erased (Fig 70). Wide experience is required for roentgenologic interpretation of

osteogenic tumors and repeated roentgenograms are often necessary to evaluate growth changes.

**Differential Diagnosis**—Periosteal and Cortical Gummata may exhibit roentgenologic findings identical with those of sarcoma. The growth rate of gumma is slower and regressive changes occur earlier. A negative Wassermann reaction should be supplemented by a provocative test before excluding lues. Enchondromas may also be confusing at times. They are usually lobulated of homogeneous consistency and do not exhibit hipping or sun ray appearance. Bone cysts are intraosteal growths although causing expansive thinning of the cortex they do not rupture the periosteum. Cysts occurring in the shafts of long bones are generally ovoid whereas those in the ends are usually trabeculated.

*Exploratory operation for biopsy is dangerous and often futile.* Although pain may be ameliorated thereby through intraperiosteal decompression growth rate is frequently accelerated. Moreover the specimen removed for pathologic examination may give little or only deceptive evidence. Irradiation is a better and safer diagnostic aid. Certain osteogenic sarcomas are particularly radiosensitive. Rapidly growing cellular types and especially Ewing's sarcoma diminish in size and become more definitely outlined. Growth rate is also lessened and pain is generally relieved promptly. Irradiation will also alleviate the pain in some resistant osteoblastic growths.

**Prognosis**—Many factors influence prognosis. Age. Tumors occurring in children and adolescents have a graver outlook than in adults. Situation. Growths situated near the trunk usually have a more hopeless prognosis those of the femoral neck, clavicle and scapula being especially unfavorable. Shoulder girdle amputation for sarcoma of the upper third of the humerus appears to offer a better prognosis than disarticulation. Growth rate. Activity of cell division and hyperchromatism are indices of rapid growth and grave prognosis. Sclerosing types and those containing an abundance of osteoblastic components evidenced clinically by slow growth offer a better prognosis.

Cell differentiation is important. Growths containing adult osteoblastic tissue offer a hopeful prognosis whereas small spindle-cell types are especially grave. Uniformity in cell structure is a favorable sign and pleomorphism an unfavorable one. However prognostication from the morphologic pattern of bone sarcoma is perhaps less reliable than gross pathologic evidence. Large tumors without metastases are more promising because virulent growth disseminates early. Encapsulation is also a favorable factor and vascularity unfavorable. (Fig. 71.) Reports from the Codman Registry indicate 80 five-year cures among 240 cases of osteogenic sarcoma (14.8 per cent).

**Treatment**—Few authentic cases of osteogenic sarcoma have been cured and most of the so-called 'cures' have occurred in gumma, giant cell tumor and osteitis fibrosa cystica. Modern therapy comprises either irradiation or surgery or a combination of both. With due respect and esteem for the untiring and conscientious efforts of its advocate, Coley's serum does not cure osteogenic sarcoma.



FIG. 71.—Vascular lytic osteogenic sarcoma.

*It is very doubtful if osteogenic sarcoma is ever cured by therapeutic irradiation.* Underexposures may stimulate growth and overdosage may produce ulceration of the soft parts and destruction of normal bone tissue, the resulting friability may also lead to fracture. When skilfully administered however radiation is of inestimable value. Pain is often definitely relieved and in cellular growths both shrinkage of the tumor and diminution of growth rate generally occur. Repeated moderate prophylactic irradiation of the lungs is indicated in all cases irrespective of the type of treatment adopted.

Resection or amputation should never be performed unless preliminary roentgenography of the chest excludes metastases. Resection of the tumor is not advisable for upon gross examination it is impossible to determine the extent of sarcomatous invasion, espe-

cially within the medullary cavity. Moreover local occurrences too often follow conservative surgery. *Early amputation or disarticulation offers the best prospect of cure.* Unfortunately in the majority of cases dissemination occurs before the onset of symptoms and the patient ultimately succumbs to metastatic recurrences.

### EWING'S SARCOMA

Ewing's sarcoma appears to be a definite pathologic entity of undetermined origin which presents a characteristic history, gross



FIG. 1-2. Ewing's sarcoma of the ulna in a child of two years before and after radiation therapy. Patient has remained well over 22 years. (Courtesy of Dr. W. H. Meyer.)

anatomy, histologic pattern and clinical course. From Fig. 69 it is evident that Ewing's tumor occurs chiefly in the shafts of long

bones and rarely in the epiphyses. Involving the shaft, the growth is usually widespread. The gross pathology is largely the combined result of aggressive growth of tumor cells, defensive reaction of the invaded bone and regressive changes in the tumor mass.

**Pathology.**—The tumor apparently arises from multiple foci in the bone-marrow of the medulla and Haversian canals. Vigorously growing foci become confluent and the growth expands in all directions. Extensive involvement often occurs at an early stage and in most instances at least one-half of the shaft is invaded when symptoms develop. The intramedullary pressure causes distention of the cortex and separation of its lamellations. This separation of the cortical layers gives the impression of thickening, and the roentgenologic picture at times resembles osteomyelitis (Fig 72). Through periosteal response, new bone may be laid down in characteristic onion-like layers. The tumor mass often resembles brain tissue both in color and consistency. With regression, cystic degeneration may result and later liquefaction of the tumor may fill the medullary cavity with material resembling pus.

Histologically, the tumor consists of small polyhedral cells with round or oval nuclei containing practically stainless cytoplasm. There is apparently a total absence of intercellular structure. The cells are uniform primitive types, capable only of producing themselves. They possess no osteoblastic potential and true bone formation never occurs. Dissemination takes place through both the blood and lymph streams and, unlike osteogenic sarcoma, involvement of neighboring lymph nodes is not unusual. The lungs and skull bones are most often invaded and the secondary growths mimic the primary pathology. At times the tumors are quite vascular.

**Incidence** —Of a total of 678 cases of bone sarcoma under Registry observation, 138 (20 per cent) are cases of Ewing's sarcoma. It is predominantly a disease of early life, the greatest incidence occurring between the ages of five and fifteen years. The tumors are rare after forty years. Occurrence is more frequent in males.

**Location** —There is a striking predilection for the long pipe bones, the epiphyses are seldom involved and joints are apparently immune. The distribution of Ewing's sarcoma is illustrated in Fig 69. When tumors occur in several bones, it is questionable whether the multiple foci are primary growths or secondary metastases.

**Symptomatology.**—There is often a history of trauma. The inception of the disease is characterized by the slow onset of mild pain and disability in one bone, accompanied by fever. In a short time the symptoms generally subside. Recurrent similar attacks, occurring at intervals of a few weeks or months, often precede the development of a tumor. Once appearing, the tumor may grow vigorously, at times, however, it partially subsides as a result of circula-



latory changes or necrosis. The exacerbations may be accompanied by a leukocytosis of 12 000 to 15 000, the relative cell differential remaining normal. The syndrome of pain, focal tenderness and fever frequently leads to an erroneous diagnosis of osteomyelitis.

The process being highly osteolytic, spontaneous fracture may develop. Ultimately the tumor assumes an aggressive phase with dissemination. Metastases occur most commonly in the lungs and skull and less often in the ribs, vertebrae and pelvis. They may develop early or be delayed for several years. Parenchymatous organs are involved infrequently.

**Diagnosis**—Trauma followed by recurrent attacks of bone pain with constitutional reaction should always suggest Ewing's sarcoma. Many cases are mistaken for osteomyelitis and if the surgeon removes only a small piece of granulation tissue for biopsy, the error may be confirmed by the pathologist. Roentgenologic evidence is invaluable. Early tumors exhibit irregular absorption of the shaft, widening of the medullary cavity, and thickening and displacement of the periosteum. Later the shaft is partially or completely destroyed (Fig. 72). Involvement is more widespread than in osteogenic sarcoma and periosteal spindle and lifting do not occur. An irregular osteolytic process with new subperiosteal bone formation may simulate osteomyelitis and wide roentgenologic

the cortex. New layers of bone which are laid down subperiosteally also become absorbed so that the periosteum appears expanded and the medullary cavity widened. The periosteum may ultimately rupture and lead to invasion of the soft parts. The involved bones are subject to deformity, infraction and pathologic fracture.

**Histogenesis**—The histogenesis of myeloma has aroused much speculation. The specific bone-marrow cells are the myelocytes, lymphocytes and mononucleated erythrocytes, and tumors believed to originate therefrom have been described as myelocytoma, lymphocytoma and erythroblastoma. The most common variety is composed of cells of the plasma type and is termed plasmacytoma. The growths have repeatedly been confused with Ewing's sarcoma.

**Diagnosis**—Myeloma is seldom recognized clinically until extensive involvement has produced a multiplicity of lesions. Pain is not a dominant feature. Early generalization through metastases especially to other bones is quite characteristic. Secondary deposits in the lungs or other parenchymatous organs are less common. Following dissemination, pyrexia and rapid emaciation ensue.

Roentgenologically the tumors appear as multiple circumscribed areas of diminished density. The medullary cavity may be expanded and the cortex thinned and navi. Radiography of the entire skeleton generally reveals multiple metastases. Bence Jones protein in the urine occurs in about one-half the cases but is not pathognomonic.

**Treatment**—Myelomas are very sensitive to both radium and high voltage roentgen rays. Repeated irradiation usually causes shrinkage and disappearance of the primary and metastatic growths. The therapy should be discontinued if the leukopenia reaches 2500. Cases have been recorded in which the condition has been held in abeyance for over ten years. In rare instances where the disease is limited to a single long bone amputation may be elected. Death ultimately occurs from recurrence and generalized dissemination.

## UNCLASSIFIED SARCOMA

Excepting very rare atypical tumors, the two pathologies which have a typical anatomic structure and clinical behavior are angioendothelioma and periosteal fibrosarcoma.

**Angioendothelioma**—Angioendotheliomas are malignant tumors which originate from the endothelium of the osseous vascular system. They are exceedingly rare and have frequently been confused with metastatic bone carcinoma. Clinically they resemble osteogenic sarcoma. Dissemination occurs through the blood or lymph streams and death generally results from pulmonary metastases. Diagnosis rests upon histologic examination.

The treatment of angioendotheliomas is apparently futile. Irradi-

ation is ineffective and amputation offers little prospect of cure as the growths often have a multiple origin and metastasize early.

**Periosteal Fibrosarcoma (Extraperiosteal Sarcoma)**—Although intimately related to the periosteum through attachment of their capsule thereto periosteal fibrosarcomas are entirely extracortical and do not invade bone. They apparently develop from the periosteal fascicular layer and 'extraperiosteal' is a more descriptive terminology. Being of fibrous tissue genesis, the tumors are incapable of producing osseous tissue.

**Incidence**—The tumors are of rare incidence being decidedly less common than fascial sarcomas with which they are frequently confused. They occur at all ages and may be congenital. Developing most often at points of tendon insertion, the growths exhibit a definite gross and microscopic pathology, roentgenologic pattern and clinical course.

**Pathology**—Periosteal fibrosarcomas remain encapsulated for many months and are often connected with the periosteum by a broad base. They are firm in consistency and upon section white and glistening. The growing mass pushes aside the soft parts and occasionally causes pressure erosion of the underlying bone. At times regressive changes produce softening and cystic degeneration. Rupture of the capsule with tumor invasion of the surrounding tissues may ultimately occur. Metastatic dissemination is a late manifestation.

Histologically, the tumors are composed of spindle cells which occur in bundles lying in an abundant fibrous matrix. The cells are generally of large type and pleomorphism is uncommon. Intercellular material predominates and vascularity is slight. The histologic pattern indicates only mild malignancy.

**Symptomatology**—The tumors have a slow growth rate and require many months to develop a moderate sized mass. Pain is not a dominant factor and mild aching or local discomfort is more commonly the first symptom. Although attached to the periosteum, the growths often appear freely movable and give the impression of being situated in the soft parts.

**Diagnosis**—The similarity to fascial sarcoma is often striking, even at operation. The differential diagnosis from osteogenic sarcoma is less difficult although roentgenologically the bone shaft may exhibit slight erosion, the tumor never surrounds the bone but appears as a faint shadow on one side of the shaft. Moreover the periosteum does not react to the tumor's presence and periosteal spindle and lifting do not occur.

**Prognosis**—The prognosis is decidedly more favorable than in osteogenic sarcoma and many reported bone sarcoma cures have been cases of periosteal fibrosarcoma. Although encapsulated tumors offer the best prognosis, extensive growths are not hopeless.

if metastases have not occurred. Rapidly growing tumors are least favorable. Recurrences following incomplete surgical removal exhibit increased malignancy.

**Treatment**—In order to effect a permanent cure radical excision of the tumor with its periosteal attachment is absolutely necessary. Remaining remnants of the latter are a common cause of recurrence. Prophylactic roentgen therapy should also be employed postoperatively. Owing to the frequency of local recurrences many surgeons advocate amputation.

### METASTATIC BONE TUMORS

Metastatic bone tumors occur commonly in carcinoma, sarcoma and hypernephroma and infrequently in Hodgkin's disease and lymphosarcoma. (Leukemia and allied blood disturbances may cause changes in bone marrow which simulate metastases.)

Carcinoma, the dominant malignancy of adult life, rarely occurs in childhood. The secondary bone deposits usually develop in the region of the nutrient artery and produce areas of rarefaction without evidence of new bone formation. Growth occurs in all directions and with destruction of the cortex pathologic fracture may result. The ribs, spine, skull, humerus and femur are the bones usually involved; the forearms and legs rarely.

Sarcoma metastasizes predominantly in the lungs. At times, however, minute tumor emboli filter through the pulmonary vessels and lodge in the bone-marrow. Such deposits occur most often in Ewing's sarcoma and in myeloma, less frequently in osteogenic sarcoma and rarely in primary visceral sarcoma. Roentgenologically the metastases mimic primary medullary growths.

Hypernephroma, essentially a pathology of middle life, rarely develops before puberty. Metastases occur through the veins to the lungs, liver or bones, especially the skull, humerus and femur.

In Hodgkin's disease and lymphosarcoma the secondary bone growths affect chiefly the bone-marrow, cortical and periosteal involvement being very uncommon. At times the lesions cause severe pain. Therapeutic irradiation generally produces regression and symptomatic relief.

**Treatment**—The treatment of secondary malignancy of bone is palliative. Support and immobilization should be employed for weakened bones and fractures. Pain may be alleviated by irradiation, opiates, nerve block, rhizotomy or chordotomy.

## PART IV

# SURGERY OF THE HEAD

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### CHAPTER XX

#### SURGERY OF THE VAULT

**Congenital Malformations** — The various varieties of cephalocele are discussed in the chapter on Neurologic Surgery. They must be distinguished from dermoid cysts, cephalematoma, cephalhydrocele and angioma. In cases of doubt the gap in the skull exhibited roentgenologically is diagnostic.

**Birth Injuries** — These comprise chiefly cephalematoma, intra cranial hemorrhage and fracture of the skull.

**Cephalematoma** — The pathology consists of an encysted extravasation of blood between the cranial bones and the periosteum. It is most commonly limited to one bone, particularly the right parietal, but at times the extravasation passes across the mid line to the opposite side. The condition generally appears first on the second or third day and increases in size for several days following. A bony ridge often develops about its circumference and in some instances a thin layer of bone forms over the whole surface and imparts a ping pong crackle on palpation. The entire mass gradually disappears, the bony ridge being the last to be absorbed.

**Diagnosis** — Cephalematoma must be differentiated from caput succedaneum, depressed fracture of the skull and cephalhydrocele. The first condition is a subcutaneous infiltration of that portion of the scalp corresponding to the center of the birth canal. The swelling is soft and diffuse and disappears in a few days. A depressed fracture may evidence indentation of the bony edges or be exhibited roentgenologically. In rare cases of fracture of the vault complicated by rupture of the dura, cephalhydrocele may develop as a fluctuant swelling of variable size which may be partly reduced by pressure.

**Treatment** — Careful cleansing of the overlying scalp is all that is required in the average case of cephalematoma. Pressure is useless and aspiration is seldom indicated. Abrasions in the vicinity should be carefully sterilized and if infection develops prompt incision and drainage are indicated.

**Intracranial Hemorrhage and Fracture of the Skull**—Both conditions are discussed in Chapter L

**Bruises and Lacerations**—These are usually due to forceps trauma. They require the same treatment as similar wounds elsewhere. Serious cases should be roentgenographed to exclude the possibility of fracture.

**Hematoma**—Hemorrhage beneath the periosteum may mimic a depressed fracture. The raised edge above the soft, apparently depressed center can usually be indented by pressure, in doubtful cases a roentgenogram will exclude fracture.

**Furunculosis and Cellulitis**—Both conditions occur commonly in poorly nourished infants. Furunculosis may be treated by daily cleansing the scalp with Castile soap water and applying 2 per cent ammoniated mercurial ointment. Pressure should be avoided. When pus appears the furuncle should be incised with a sharp pointed bistoury. Drainage is unnecessary.

Cellulitis of the scalp may be treated with wet compresses of boric acid solution or 10 per cent alcohol. Incision and drainage should be performed promptly if fluctuation develops. In extensive cases multiple small incisions are preferable to one large incision. (A low grade cellulitis of the scalp may pass unrecognized for several days unless the infant is carefully examined.) Attention to the child's nutrition is very important and in protracted cases blood transfusion is often of great benefit.

**Erysipelas** Invasion of the scalp by erysipelas is at times puzzling. The constitutional reaction is generally severe but the characteristic redness and line of demarcation often do not appear until the hair line is passed. Most cases recover. Cures following the oral or intravenous administration of sulfanilamide are often swift and spectacular.

**Pneumatocele** In rare instances air collects under the periosteum from inflammation of the cortex over air-containing cells. The infected part should be incised and the area probed with iodoform gauze.

**Cysts**—Sebaceous and dermoid cysts are discussed elsewhere. The former rarely occur before puberty and the latter appear at embryonic fusion sites. (Refer to Chapter IX.)

**Nevi**—Vascular tumors including cirroid aneurysm are discussed in the section on Hemangiomas.

**Benign Tumors**—These comprise lipoma and fibroma. Both are rare.

**Scalp Wounds** Slight wounds may be sterilized by applying full strength tincture of iodine followed by a gauze dressing held in place by collodion on cotton. In the case of extensive wounds the parts should be shaved and thoroughly cleansed with soap and water followed by the application of full strength tincture of iodine. All

devitalized tissue should be débrided. The skull is then explored for possible fracture, following which the soft parts are approximated with dermal suture. A rubber tissue or elastic band drain is advisable for forty-eight hours. The excessive vascularity of the scalp accounts for the large percentage of healing per primum in potentially infected cases.

**Avulsion**—The scalp may be partially or completely avulsed. In partial types the flap should be carefully cleansed and replaced by suture. Its viability will depend upon circulatory competence.

Cases of complete avulsion require meticulous care. The patient should be taken to the operating room and the parts thoroughly sterilized with full strength tincture of iodine, followed by 70 per cent alcohol, all devitalized portions of the remaining scalp should be carefully débrided and hemostasis secured. The wound should then be treated by the Carrel-Dakin technic until both smears and cultures indicate sterility before skin grafting is attempted. Not infrequently superficial portions of the skull bones exfoliate before the granulations can be grafted. The recommended procedure of boring holes into the diploe to stimulate granulating formation is ill advised. The skull bones of children contain little diploic tissue and the practice is definitely dangerous. In the author's experience, pinch grafts have been more successful than Thiersch or full thickness types. Occasionally sliding grafts may be employed from the healthy pericranium.

## CHAPTER XVI

### CONGENITAL CLEFT LIP AND PALATE

By HAROLD S. VAUGHAN, M.D., D.D.S., F.A.C.S.

THE term congenital cleft lip and palate denotes an early embryonic failure of union between the separate processes which form the lip, alveolar border hard and soft palate. There may be any degree of deformity ranging from a bifid uvula, or a notched vermilion border of the lip, to a failure of all the various processes to unite, thereby producing a bilateral complete cleft lip and palate.

**Etiology**—Cleft lip or palate occurs about once in every 1200 births and is more frequent on the left side. The voluminous literature on the etiology indicates that none of the implied factors offers a satisfactory explanation. Many of them must be regarded as highly fantastic, especially those in relation to the exciting causes.

**Race**—Cleft lip and palate are relatively rare in negroes. Among white races the defects are distributed without regard to racial distinction. **Sex**—Males are more liable than females.

**Heredity**—This is a dominant factor. A very large percentage of cases reveal similar defects in other members of the family. The frequent denial of any such familial defect usually applies only to the parents and grandparents for, as a rule, they know nothing of earlier progenitors. Great difference in ages of the parents has been noted in some cases.

**Exciting Causes**—Discussion of the exciting causes is largely speculative. Maternal prenatal impressions have no basis of fact as the supposed shock has usually occurred long after fetal development has progressed beyond the point where it could be a factor of influence. The author has frequently seen twins where only one was afflicted. Defective nutrition or general weakness of the mother during early pregnancy may be a factor in delaying union. Due to the flexed position of the head, pressure of the tongue on the palate and of the mandible against the sternum has been advanced as an exciting cause by Brophy. Warnekros, studying the teeth of cleft palate cases, concludes that supernumerary teeth may be a causative factor. It would appear, however, that the factors producing the cleft should be regarded as the cause of supernumerary teeth since the tissue intended for a normal tooth follicle becomes divided by the cleft.

**Development**—In early fetal life, the primitive mouth and nasal fossa in conjunction with the forebrain and pericardium form a single cavity separated from the foregut by the pharyngeal membrane which later disappears. The maxillary process, growing inward from the lateral walls of the cavity, separates the nasal from the oral cavity. The anterior part of the palate is developed from



the fronto-nasal process which growing downward from the frontal region forms the anterior part of the nasal septum the median portion of the lip and the premaxillary bones

The olfactory pits appearing on the lower and anterior surface of the fronto-nasal process divide the process into the median and lateral processes. From each lateral angle of the median nasal processes two elevations develop, termed the globular processes. The portion between the globular processes forms the lower part of the nasal septum and the columella while the globular processes unite to form the median portion of the upper lip (the philtrum) and the premaxillary bones. The nasal alæ are formed by the lateral nasal processes.

The maxillary processes arising from the mandibular arches growing inward unite with the lateral nasal processes and the globular processes, completing the lip at about the eighth week. The palate is formed by the union of the maxillary processes posteriorly and the development and union anteriorly of the premaxillary processes with the maxillary. This union of the three portions of the palate is completed by the tenth week.

Ritchie and Staige Davis have developed the following descriptive classification for these defects

GROUP I	Prealveolar (process) cleft lip cleft process normal			
1 Unilateral	Right	{	Complete cleft extends into the nostril	
			Incomplete cleft does not extend into the nostril	
	Left	{	Complete	
			Incomplete	
2 Median (rare)		{	Complete	
			Incomplete	
3 Bilateral	Right	{	Complete	Left { Complete Incomplete
			Incomplete	
GROUP II	Postalveolar (process) cleft palate cleft process normal			
1 Soft palate		Extent in thirds		
2 Hard palate		Extent in thirds		
Situation and attachment of septum				
Note If associated with lip cleft fall in Group I form				
GROUP III	Alveolar (process) cleft follows incisive sutures			
1 Unilateral				
	Process	Right	{	Complete cleft extends through alveolar process Incomplete cleft does not extend entirely through alveolar process
	Palate		{	Unilateral—bilateral—median Complete—incomplete
	Lip		{	Unilateral bilateral—median Complete incomplete
2 Median (rare)			{	Complete Incomplete
3 Bilateral				
	Process	Right	{	Left { Complete Incomplete
	Palate	Right	{	Left { Complete Incomplete
	Lip	Right	{	Left { Complete Incomplete

**Clefts at the Angle of the Mouth** — In rare instances the cleft occurs at the angle of the mouth and the fissure may extend a variable

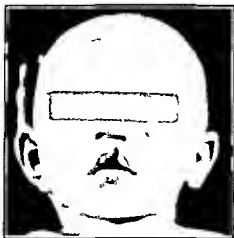


FIG 3 — Unilateral cleft lip incomplete left side with cleft palate



FIG 4 — Unilateral cleft lip incomplete left side with cleft palate

distance into the cheek at times reaching the orbit (figs 103 and 104). In extreme cases it may involve the temporal region and in a specimen from the Royal College of Surgeons quoted by Brophy, the cleft passed through the temporal region to the occiput.



through the molar region and traverse the palate obliquely leaving the central portion of the soft palate intact



FIG 77 —Bilateral cleft lip complete both sides with displacement of premaxilla and palate cleft



FIG 78 Median cleft lip complete with slight lateral clefts at the angle of the mouth on each side and oblique clefts of the palate

### ANATOMY OF THE LIPS

The lips are fleshy folds covered by the skin externally and mucous membrane internally. The mucous membrane continuous with the buccal membrane is reflected over the gums in the mid line forming the frenum labii superioris and the frenum inferioris. The margins of the lip are covered with a dry mucous membrane termed the vermillion border. Its junction with the skin is well defined and forms a continuous line a point to be emphasized in cleft lip operations. The median portion of the upper lip forms a wide shallow furrow the *philtrum* the lower border of which is known as the labial tubercle. Between the integument and mucous membrane the lip contains the superficial fasciae orbicular muscle and attachment of the facial muscles. The most important in connection with the cleft lip are the levator labii superioris alaeque nasi and the compressor narium. The submucous alveolar tissue contains the labial glands coronary vessels motor and sensory nerves.

### ANATOMY OF THE PALATE

The palate consists of two parts the hard anteriorly and the soft posteriorly. The hard palate composed of the horizontal processes of the superior maxillary and palate bones is covered on the inferior surface by a dense mucoperiosteum. Laterally it is bounded by the alveolar process. Circulation is derived from the nasopalatine

posterior palatine and accessory palatine vessels supplemented in the soft palate by branches from the facial ascending pharyngeal and tonsillar arteries. The importance of the circulation should be considered when attempting extensive operative procedures lest tissue necrosis result from destruction of the blood supply. The nerve supply is from the nasopalatine and anterior palatine nerves.

The soft palate is a musculo-membranous movable curtain attached anteriorly to the border of the hard palate while posteriorly the border is free. The following structures are contained between its two layers of mucous membrane: the two levatores palatini tensoris palati palato-pharyngeus palato-glossus and azygus uvulae muscles also an aponeurosis glandular tissue vessels and nerves. The uvula, a conical prolongation from the middle of the posterior border, consists chiefly of gland tissue and the azygus uvulae muscle.

The concave posterior margins of the palate containing the palato-pharyngeus muscles extend laterally on the posterior pharyngeal wall forming the posterior pillars of the fauces. Anteriorly, a smaller fold containing the palato-glossus muscles passes downward and forward on each side of the base of the tongue forming the anterior pillars of the fauces. The thin mucous membrane of the soft palate is covered with squamous epithelium on the buccal surface and columnar ciliated on its nasal side.

### PHYSIOLOGY OF THE PALATE

The functions of the hard palate are to separate the mouth from the nose and thus prevent the passage of food and air into the nasal cavity to aid mastication and to give quality to the voice. The function of the soft palate is to prevent food from passing into the nose through elevation and tension of the velum. In phonation and articulation the velum shuts off the nasopharynx and nasal cavities from the oropharynx thereby permitting the production of clear voice sounds.

When the velum is defective it is impossible to close off the oropharynx from the nasopharynx. In the articulation of consonants air that should be expelled through the mouth passes into the posterior nares imparting a nasal resonance to the speech. It is often noted that cleft palate patients contract the anterior nares to prevent air from passing through. A certain degree of closure of the oropharynx is obtained by the action of the superior constrictor muscles.

### PREOPERATIVE TREATMENT OF CLEFT LIP AND PALATE

As the cleft lip and palate infant is unable to take the breast or even the bottle nipple if the defect is extensive care must be taken to see that the infant is properly nourished before the operation is

considered; this responsibility should be assumed by the pediatrician. Although the repair of cleft lip may be safely performed in the new-born it is advisable to wait until the feeding has been stabilized and the baby shows a steady gain in weight, disaster may follow operation during an attempt to find a satisfactory feeding formula. It is often necessary to use a medicine dropper, or at times the Breech feeder may prove satisfactory. Roentgen-ray examination to determine the presence of an enlarged thymus should be a preoperative routine.

It may require several weeks before the infant is in satisfactory condition for operation. If the cleft between the alveolar borders is wide with considerable displacement of the premaxilla, operation should not be too long delayed as the bones become less pliable as calcification takes place. The first objective to be attained in a case of complete cleft lip and palate is to restore the correct anatomic relation of the premaxilla, alveolar border, lip, nasal ala and columella. The plastic closure of the hard and soft palate should not be attempted before eighteen months and the author obtains better results between the second or third year, or even later.

When operating for cleft lip and palate, the Rose position is preferred. The anesthetist sits at the left and ether vapor is administered through a nasal catheter or a curved metal tube hooked in the angle of the mouth. A capable assistant attends to the suctionage, sponging and passing of instruments, a nurse prepares the sutures.

## REPAIR OF ALVEOLAR BORDER AND UNILATERAL CLEFT LIP.

In unilateral complete lip, Blair and many others advise repair of the lip without regard to the alveolar border cleft. Although the latter will close in a large proportion of cases there are many others in which the alveolar cleft persists, and the large opening under the lip, extending into the floor of the nose, is difficult to close. It is therefore advisable to so approximate the alveolar border that the opening into the nasal floor can be readily repaired.

If the lip is repaired before the third month and the cleft is not too wide, the alveolar border can be approximated by digital pressure, the action of the repaired lip further aids the approximation. However, if the alveolar cleft is wide, it is necessary to hold the approximation by a 20- or 22-gauge silver wire. This is passed through the canine region at the level of the floor of the nose, so as to avoid the tooth follicles. This point cannot be overemphasized. The wire is buried under the mucosa, and the two ends, emerging at the median line, are twisted together with sufficient tension to hold the approximation obtained by digital manipulation.

The wire should never be used to force approximation as it will cut into the tissues. The next step is to repair the lip (Figs 79 and 80).

A great variety of operations have been suggested for the repair of cleft lip but as Blair has so truly stated the operator will obtain better results by developing a technic with one particular procedure.



FIG. 79—Indicates the sulcus wire buried deeply enough to hold the tooth follicles in the approximation of a wide alveolar cleft.



FIG. 80—Indicates sulcus wire tightened to hold alveolar borders after approximation by digital pressure.

**The Nelaton and Rose Methods**—The time-honored Nelaton operation for the correction of a notched lip is rarely satisfactory (Fig. 81). It is difficult to obtain a good vermillion border; moreover, cases usually show a separation of muscle above the notch which may extend into the nose. The Rose operation can be performed with



FIG. 81—The Nelaton method.

satisfactory results. Useful tissue, however, is sacrificed which tends to reduce the vermillion border. A curved incision is made on each side of the cleft from above downward, the knife passing through the vermillion border at about its widest part. The degree of curvature in the incision depends upon the required length for the

lip the curved line becomes straight when the lip is pulled down and when sutured there is a redundancy of vermillion border at the point of union which prevents a notched border (Fig 82) The Rose method is not suitable for a bilateral cleft as there is too much elongation of the lip (Fig 83)



FIG 82 —The Rose method for unilateral cleft lip

**The Owen Operation** —The Owen operation may be quite satisfactory for a complete cleft lip where there is a well developed vermillion border on the median side of the cleft. The vermillion border is removed from the alar side of the cleft beginning at the columellar side of the cleft an oblique incision is passed downward about half way between the vermillion border and the nose where it passes transversely forming an obtuse angle. It is then sutured as shown in Fig 84. In selected cases this method produces a loose lip with abundant tissue and an ample vermillion border.

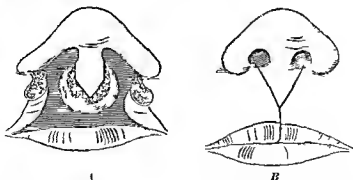


FIG 83 —Rose method for bilateral cleft lip. A Bilateral cleft showing incisions. B Operation complete upper lip elongated.

As previously stated better results can be obtained by developing a procedure applicable to all cases. The author has for several years depended upon a modified Virault operation somewhat after the plan followed by Blair to whom he is indebted.

**Author's Operation** —The ala is separated from its attachment to the alveolar border and the lip is very thoroughly undermined including the columella. Calipers are used to measure the length

of the lip from the margin of the ala on the normal side to the vermillion border. The same distance is marked from the columellar margin of the cleft to the junction of skin and vermillion border.

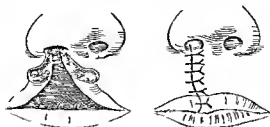


FIG. 84 —The Owen method for repair of unilateral cleft lip

The vermillion border is separated along the skin junction of the cleft from the columellar end down to the point marked by the calipers. The calipers are closed to one-half this distance and a triangle is laid out on the ala side from a point just below the ala (Fig. 85). The sides of the triangle 1-3, 1-2 and 2-3 is removed

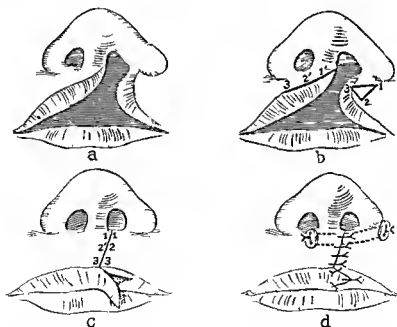


FIG. 85 a complete cleft lip 1 plan for skin incisions c adjustment of flaps d lip sutured and wire tension sutures with key buttons adjusted

A buried suture of 000 chromic gut is used to approximate the ala to its correct position so that it joins I to I'. As the ala is turned into position, the adjacent tissue is used to build up the floor of the



nose The skin is approximated with fine dermal or silver wire so that 2 joins 2 and 3 joins 3 care being taken to have the skin bor-



FIG 86 — Unilateral cleft lip incomplete with cleft palate

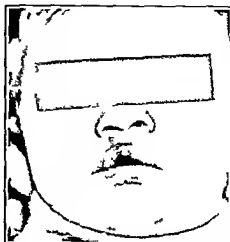


FIG 87 After repair of lip and correct on of nasal ala

der continuous The alar side of the vermillion border is split and the necessary amount of surplus tissue from the opposite side is fitted into it and sutured Deep sutures of heavy dermal which



FIG 88 — Unilateral cleft lip incomplete with flattened and displaced ala



FIG 89 — After repair of lip and adjustment of ala Photograph taken at time sutures are removed

approximate the muscle are inserted on the under surface The ala is supported by tension sutures tied down on lead buttons

A Logan bow is then applied for relief of tension on the suture line. Figs. 86 to 93 illustrate cases after repair by the above method.

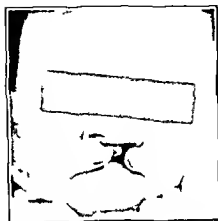


FIG. 90 — Unilateral cleft lip incomplete with cleft palate



FIG. 91 — After repair of lip and adjustment of nasal ala



The favorite method has been to excise a V shaped section from the vomer then by forcible pressure the premaxilla is carried backward in relation to the alveolar border. This procedure rotates the bone on its transverse axis so that the teeth are directed backward. Other surgeons advise that a square section be removed from the vomer so that the premaxilla can be slid backward as a drawer would be closed. Even in moderate deformities complete separation of the premaxilla from the vomer takes place by either of these methods. It tends to drift out of place and complete fixation is difficult. Moreover the blood supply is impaired and the premaxilla rarely develops sufficiently to carry the incisor teeth. The end result is an underdeveloped premaxilla which does not provide sufficient bone support to maintain the contour of the lip and the latter becomes progressively more flattened as the child develops.

In any operative procedure on the premaxilla it is very important to control the process so that it cannot become displaced. Otherwise it will fail to keep developmental pace with the adjacent structures.



FIG 94—Shows the method of cutting obliquely through the vomer near its posterior end so that the premaxilla can be adjusted without damage to the blood supply.

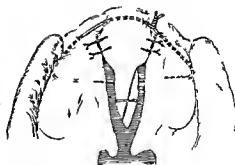


FIG 95 Shows the premaxilla in position. It is safer to hold the premaxilla with a buried silver wire which passes through the maxilla and anteriorly to the premaxilla thus preventing forward displacement.

**Treatment of Projecting Premaxilla**—The author prefers to maintain support of the lip by retaining a rigid premaxilla for this purpose. If the projection is not too pronounced this can be obtained by forcing the premaxilla backward as far as possible by digital pressure sufficient to permit closure of the lip cleft. Splitting the vomer is often of help before making pressure. When necessary a silver wire is passed through the maxilla above the alveolus sufficiently high to avoid the tooth follicles. The procedure should never be undertaken unless the operator uses a technic that

will avoid the tooth follicles. If, however, the premaxillary projection is so extreme that the lip cannot be repaired over it, the method of Bardeleben should be employed. An incision is made along the lower border of the septum and the mucoperiosteum elevated, an oblique cut is made through the septum near the posterior end extending well up the septum so that the premaxilla can be forced backward without rotation. It is then held in position by a silver wire suture passed through the overlapped sections (Figs 94 and 95). The objection to cutting through the septum close to the premaxilla is that it interferes with the blood supply so that the process will not develop sufficiently to support the lip.

If the premaxilla is in contact with the alveolar borders, the latter are freshened and united with the premaxilla on each side. This is not done however unless contact obtains, as it is far better from the standpoint of future development to obtain a proper relation between the upper and lower dental arches. The upper arch should be larger so that the lower teeth, when erupted, will not be anterior to the upper.

### THE REPAIR OF BILATERAL CLEFT LIP

Where forcible adjustment of the premaxilla is necessary, the lip operation should be postponed for a few days until the baby has recovered from the first procedure.

In nearly all operations advised for the repair of bilateral cleft lip, the vermillion border is removed from the prolabium, and the alar borders of the cleft are united around the denuded prolabium. This plan tends to produce an elongated lip which is, in most cases, out of harmony with the face. This is especially true of the Rose operation, indicated by Fig 82.

Insufficient consideration is given to the fact that the prolabium furnishes enough tissue for the central portion of the lip, if given an opportunity to develop. With this end in view, the author plans his operation with the object of retaining the vermillion border at the lower end of the prolabium and supplementing it with the vermillion border from the alar sides of the cleft.

**Author's Operation for Bilateral Cleft Lip**—The entire central portion of the lip is conserved. Even though scant in size, this tissue will develop surprisingly, and produce a lip of the proper length. In most cases, even when later revision is necessary, a far better result obtains than from revision of an unsightly long lip.

An incision is made along both borders of the prolabium from above downward, separating the vermillion border from the skin down to the lower border, the central portion of the vermillion border being left intact. The prolabium is measured with calipers and the calipers set to one-half this distance. Points are made with

calipers in the skin just below the alæ on each side and at the vermillion border, triangles are laid out represented by 1-2 3, in the illustration (Fig 96) The full thickness of the lip is cut through from 1 to 3 and from 2 to 3, the cut from 1 to 2 includes only the skin This triangle of skin only is then removed The same procedure is followed on the opposite side The alæ are separated from the alveolar borders and thoroughly undermined, an incision is then made across the remaining vermillion border on the prolabium The ala is turned in and point 1 is approximated and sutured to 1', using a buried suture of chromic gut, the redundant tissue is utilized to form the nasal floor, 2 is joined to 2', and 3 is

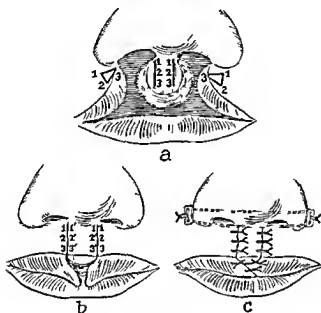


FIG 96 —Shows the method used by the author in the repair of bilateral cleft lip using the principle developed by Blair

joined to 3' This is repeated on the opposite side The free vermillion border from each side is fitted into the incised vermillion border across the lower end of the prolabium, and sutured The alæ are supported by tension sutures which pass through lead buttons The skin sutures are of either fine dermal or fine silver wire which ties easily

Deep sutures of heavier dermal are used on the under surface and serve to approximate the muscle The Logan bow is then applied and the suture line covered with vaseline or zinc oxide ointment as a protection from nasal secretions

In cases where there is unusual tension on the suture lines, the Logan bow may not furnish the necessary support, it is then safer

to apply adhesive plaster on each side of the face, two silver sutures being passed through the adhesive on one side under the lip and through the adhesive on the opposite side. The wires are then twisted over lead buttons to the required tension, the adhesive plaster prevents the silver sutures from cutting through the tissues. The skin sutures are removed on the fifth or sixth day, the deep sutures on the seventh.

**Corrections To Be Made Later** —The prolabium is usually bound down to the premaxilla thereby interfering with free mobility of the lip. This condition is later corrected by freely separating the lip from the premaxilla. A stent is then moulded to the incised area, a split thickness skin graft is wrapped around the stent which is then replaced and held in position with sutures for about ten days. Some prefer a fat fascial transplant, after the method of Ferris-Smith.

Following the replacement of a prominent premaxilla and repair of a bilateral cleft, the tip of the nose is generally drawn down and flattened. This is later corrected by the following procedure. An incision is made in the columella at the anterior end of the alar cartilage, down to the alar cartilage, the incision is then extended to the median line at the mid-portion of the lip. This is repeated on the opposite side forming a V shaped incision. The tissue is elevated from the columellar cartilage and lifted upward and forward. The incision is then sutured as a Y, raising the tip upward and forward.

**Postoperative Care** —The child should be wrapped warmly in bed and turned on its side so blood or mucus will drain out of the mouth. The foot of the bed should be elevated. In a lip operation on a young baby care should be taken that an airway is open for proper breathing for at times the lips become compressed in a valve-like action and breathing is interfered with, this can be remedied by the insertion of a rubber tube in one nostril. Feedings are resumed three hours after operation. Elbow splints or cardboard cuffs prevent the baby from putting its fingers in the mouth or disturbing the lip suture line.

Constipation is avoided by mild laxatives or a daily enema. Sutures are removed in from five to seven days, the tension stays remaining a day or two longer.

### SURGICAL REPAIR OF CLEFT PALATE

The term cleft palate denotes congenital lack of union of any portion of the soft palate, hard palate, or alveolar border. (See classification, page 203.)

**Time to Operate** —The operation for the plastic repair of a hard and soft palate cleft should not be performed before eighteen months and better results and a lower mortality are obtained by waiting

until the second or third year, or even later. Several considerations favor operating at this time—as the teeth erupt, the alveolar process develops and the palate becomes more arched, thereby providing more tissue to close the cleft, moreover the palate tissues are then more developed, thicker and less friable. It was formerly considered necessary to close a cleft palate before the child commenced to talk, or before the characteristic cleft palate speech became established. This, however, is a wrong premise as cleft palate speech is dependent upon the length of the palate and whether it will function so as to prevent air from passing into the nose. In previously operated cases of older children and adults presenting a shortened palate with thickened and underdeveloped palato-pharyngeus muscles, marked speech improvement may be obtained by using the posterior pillars to lengthen the palate.

Before operation the general health and physical condition of the child should be favorable, also free of nasal and oral infections. The winter months are not elected for operation as upper respiratory infections are more prevalent and the bacterial flora more varied and virulent. Such factors predispose to ulceration of the raw surfaces with resultant breakdown of the suture line.

Favorable results depend upon several important factors: (1) Recognition and conservation of the blood supply in the preparation of flaps and elevated soft tissues, (2) lateral incisions sufficiently long to permit approximation of the suture line without tension, (3) supplementary support of the suture line by immobilization of the soft palate and relief of tension, (4) support of the suture line in the hard palate, and relief from tongue pressure when necessary.

The basic principle of the von Langenbeck operation gives the best results. This comprises elevation of the tissues from the palatal vault, separation of the palate aponeurosis at its attachment to the palate bone, paring the borders of the cleft, and suture in the median line. Modification is necessary to suit the individual case.

**Surgical Treatment of Cleft Palate Involving the Soft Palate and Extending Into the Hard Palate**—After the borders of the cleft have been freshened, the soft tissues are elevated from the hard palate by blunt dissection. The lateral flaps then drop and tend to approximate in the median line, especially if the arch of the palate is high. They may be further approximated by separating the aponeurosis which attaches the soft palate to the palatal bone. Ordinarily this procedure suffices for approximation when the cleft is narrow. Wider clefts require lateral incisions to relieve tension on the median line. The lateral incisions should always be made close to the teeth so as to traverse the palate external to the palatine arteries, and be extended well around the maxillary tuberosity to

relieve tension at the junction of the hard and soft palate, the site where non-union and perforation are likely to occur



FIG 97

FIG 97 — Bilateral cleft lip, incomplete

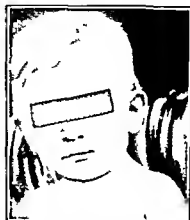


FIG 98

FIG 98 — After repair the prolabium was utilized for the skin portion of the median part of the lip. The vermilion border was supplemented from the external portions producing the necessary eversion



FIG 99 — Bilateral complete cleft lip with anterior displacement of premaxilla



FIG 100 — After replacement of premaxilla and repair of lip

The soft palate is closed with fine dermal suture, using a straight needle to produce the same contact on the nasal surface. A second row of sutures is placed on the nasal surface of the uvula and the posterior portion of the palate, to supplement those on the oral surface



In addition to the ordinary interrupted stitch mattress sutures are also employed in the hard palate in order to obtain a lapped approximation instead of an edge-to-edge one. Lateral incisions



FIG. 101 — Shows cleft extending from the mouth into the cheek on each



FIG. 102 — Shows the clefts extending obliquely through the molar region of the hard and soft palate



FIG. 103 — Shows a cleft on the right side extending into the temporal region



FIG. 104 — Shows a cleft on the left side extending into the eye

are also made on each side external to the palate muscles for the insertion of the author's tension relief guards.

Support of the hard palate suture line is advisable in wide clefts where the highly arched vault of the palate has been so reduced

as to encroach upon the space occupied by the tongue. In such instance the upward pressure of the tongue may tend to open the suture line. This is obviated by inserting either a narrow lead



FIG 105 Shows oblique clefts extending through the molar region hard and soft palate



FIG 106 — After the first stage of repair on each side

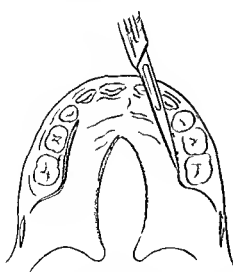


FIG 107

FIG 107 Indicating a wide cleft through the hard and soft palate and showing the lateral incisions and the incisions for the author's guards which are used to prevent the tension relief wire from cutting into the tissues. The same incisions are used for the Mackenty lead ribbon.

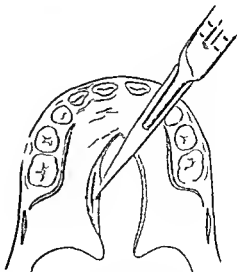


FIG 108

FIG 108 Incision along the septal mucosa prior to the elevation of the soft tissue from the bony palate

ribbon or a silver wire supported by the author's tension relief guards. Figs 107 to 119 illustrate the author's procedure for the von Langenbeck operation.

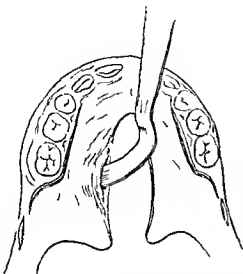


FIG 109

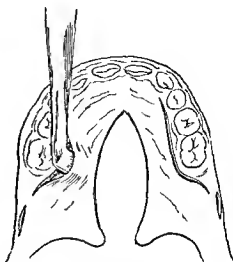


FIG 110

FIG 109 —Elevating the palate tissues outward from the border of the cleft

FIG 110 —Elevating the palate tissues inward from the lateral incision with care rupture of the palatine vessels can usually be avoided

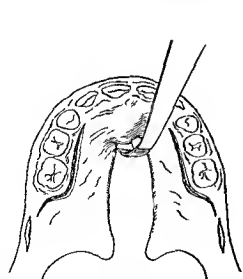


FIG 111

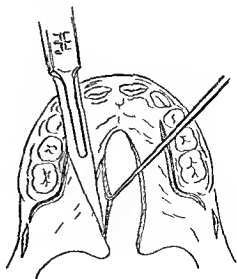


FIG 112

FIG 111 —Elevation of the tissues from the palate anteriorly

FIG 112 —Excising a thin section of mucosa from the cleft border

A soft cleft palate rarely contains enough tissue after repair to reach the posterior wall of the pharynx and thereby correct the nasal quality of the voice which results from air passing into the

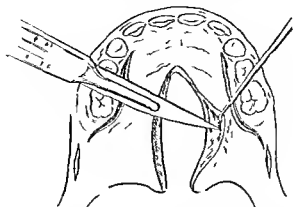


FIG 113 —Incising the aponeurosis from the posterior border of the palate bone

nose in the articulation of consonants. The ultimate goal is to secure normal speech, and the various operations for closure of the palate cleft fail to attain this end in most cases. Experienced operators, beginning with Passavant in 1862, have tried various methods to lengthen the palate or constrict the pharynx and thus overcome

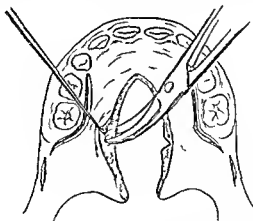


FIG 114 —Completing the incision of the palate aponeurosis with curved scissors  
The borders of the cleft can then be approximated

the anatomic defect. Interest in the subject has been further stimulated by the article of Dorrance on "Lengthening the Soft Palate in Cleft Palate Operation" (1925).

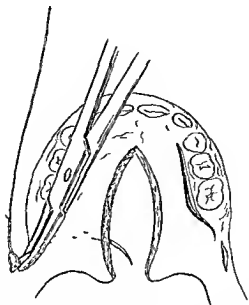


FIG. 115.—The insertion of a silver or bronze wire suture for the relief of tension

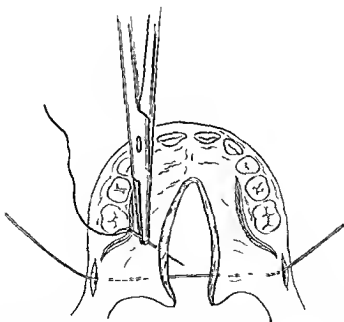


FIG. 116.—Inserting the soft palate sutures. A straight needle is used with 00 dermal. The needle forms an obtuse angle to the holder for the first insertion and for the return on the opposite side the needle forms an acute angle to the holder. This produces a broad surface contact equal to an additional row of sutures on the nasal side.

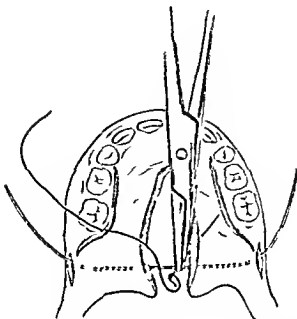


FIG. 11" —Inserting the straight needle on the opposite side to the acute angle of the needle to the holder

The author's technic for lengthening the palate when the cleft extends through the soft and into the hard palate (Fig 120 *a*) consists of a two-stage procedure. The first closes the cleft by the

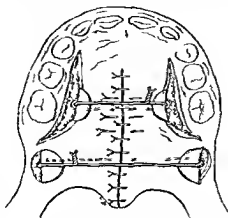


FIG 119 Shows the operation completed with ten wires tightened and guards in position

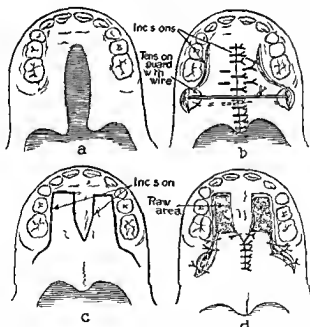


FIG 120 — *a* Illustrates a cleft extending through the soft and hard palate to the premaxilla. This cleft is too extensive for the Dorrance method. *b* The palate after closure. *c* The incision used by the author to elongate the palate in cases where the cleft extends through the palate to the premaxilla. *d* The elongated palate sutured into position.

method described (Fig 120 *b*) the second may be performed any time after full recovery from the first operation, preferably several weeks or months later.

**Second stage Operation to Lengthen the Palate**—With the patient in the Rose position a U shaped incision is made on each side of the palate leaving a central section of tissue covering the original cleft in the hard palate. The internal arm of each U meets in the median line as far back as the posterior border of the palate bone. The external arms of the incisions extend close to the teeth around the tuberosities and external to the palate muscles, forming a double U (Fig 120 c)

The flaps are then elevated and freed from their bony attachments. The hamular process is separated, releasing the tendon of the tensor palati muscle. If the palate is not extremely short, it can then be carried backward to reach the posterior pharyngeal wall.

The lateral flaps are sutured to the apical portion of the median section of the palate, as illustrated in Fig 120 d. The lateral incisions are then sutured posterior to the tuberosities. The raw surfaces are covered with iodoform gauze held in position by wires which cross the palate and are attached to the teeth (Fig 121). This method permits of a much greater lengthening of the palate without the danger of producing an anterior opening in the hard palate.

FIG 121 Illustrates the use of wires attached to the teeth to hold the iodoform gauze packing against the raw surfaces

Figs 122 a to 122 d show a 'push back' technique with the incision extending around the lingual surfaces of the teeth as in the method of Dorrance. With this incision the distance the palate can be pushed back is limited by the degree of cleft extension into the hard palate if an anterior perforation is to be avoided. Unless the cleft is limited to the posterior portion of the soft palate the author always repairs the cleft sometime prior to the operation for elongating the palate.

The operation for lengthening the palate has been limited to incomplete cleft palate. Heretofore when a Dorrance "push back" operation was performed on a complete cleft the patient had to wear a prosthetic appliance to cover the anterior opening. In the author's procedure all types of cleft palate can be lengthened whether complete or incomplete.

### COMPLETE CLEFT, ALVEOLAR PROCESS AND PALATE (GROUPS II AND III)

With complete cleft palate when the cleft is wide (Fig 123) it is best to close the palate by a two stage operation. The same operative procedure is carried out as previously described for an incomplete cleft. The soft palate is closed including as much of the



hard palate as can be utilized without injury to the blood supply of the anterior flaps. An additional lead ribbon or tension relief guard may be necessary as a protection against tongue pressure (Fig 124 a)

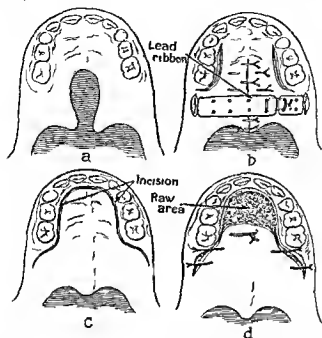


FIG 122 a Illustrates a cleft extending a short distance into the hard palate and suitable for a Dorrance push back operation b After repair of the cleft A MacKenty lead ribbon is used in this case for relief of tension on the suture line The author allows several weeks or months to elapse before operating to elongate the palate c The Dorrance incision for a push back operation d The palate elongated and held in position by a silver wire through the bone



FIG 123 —Illustrates a wide complete cleft through the alveolar border, hard and soft palates suitable for the author's operation

After the child has convalesced from the effects of the first operation the anterior portion of the cleft, including the alveolar defect, may be closed. The best procedure is to freshen the borders of the cleft on each side. An incision is then made beginning with

the premaxilla side of the cleft close to the teeth and followed around to the maxillary tuberosity on the same side. The flap thus formed is entirely elevated from the bone beginning with the cleft border. The tissues are then elevated along the opposite border of the cleft and the edge of the free flap is carried under this raised border and sutured into position thus covering the defect (Fig 124 b).

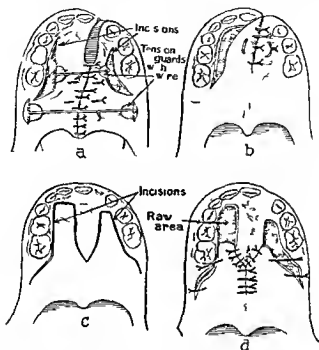


FIG 124 a Illustrates the case shown in Fig 193 after closure of the soft palate and part of the hard palate. b After closure of the anterior cleft by sliding flap method. c The author's incision to elongate the palate after the two-stage repair of a complete cleft. d The palate elongated and sutured to the posterior end of the tissue covering the cleft.

The author has observed that the palate is usually longer following repair of a complete than an incomplete cleft, but if the desired speech improvement has not been obtained the palate can be elongated by the following procedure. The outline of the cleft through the hard palate is obtained by needle punctures through the overlying tissues. A double U incision is made similar to the one described for an incomplete cleft but enough tissue must be left to cover the original cleft, there being less tissue available anteriorly on the cleft side (Fig 124 c). The soft tissues within the U on each side are elevated and separated from their bony attachments. The hamular processes are fractured with a small chisel releasing the tensor palati on each side. The nasal aponeurosis is left attached to the posterior margin of the palatal bone externally.

to correspond to the raw surface left when the palate is elongated posteriorly. Careful undermining is necessary to permit this elongation. If the palate is not too short it can then be moved backward to touch the posterior pharyngeal wall (Fig 124 d). To be certain that the blood supply is adequate the flaps are then returned to their original location and held by a few retention sutures. At the next operation four or five days later the flaps are again elevated, carried backward and sutured to the posterior end of the tissue covering the cleft in the hard palate. The open central portion and sides are sutured leaving raw surfaces anteriorly to be filled in by granulation. This method extends the benefits of palate elongation to a large group of cases that cannot be aided by the Dorrance push back operation.

**Postoperative Care**—The child should be put to bed face downward so that any blood can run out of the mouth. The foot of the bed should be elevated. Liquids including milk may be given after a few hours and an abundant fluid intake should be encouraged. Water should also be given after each feeding to prevent food lodging in the mouth. If necessary sedatives may be administered also mild laxatives. Elbow splints are employed to keep the child's hands out of the mouth. Iodoform dressings are changed every other day and sutures are removed under primary anesthesia about the eighth or tenth. Speech training is of value in a large percentage of cases though much more is accomplished if commenced between the fifth and sixth year.

**The Brophy Palate Operation**—This procedure has lost favor for the following reasons: considerable mortality; many of the tooth follicles are destroyed; the dental arch remains contracted and extreme malocclusion of the teeth frequently results. Under present methods complete palate clefts can be readily repaired without damage to the developing teeth.

**The Lane Palate Operation**—This method is rarely used at the present time and is open to serious objections. Even when the closure is successful the palate shortened by cicatricial contraction becomes stiff and rigid. Speech results are poor and from an anatomic and physiologic standpoint the operation should never be attempted.

## NON-SURGICAL TREATMENT OF CLEFT PALATE

It is possible to surgically close any congenital cleft of the hard and soft palate. However there are some cases in which infection has destroyed so much tissue that further surgical interference can not be considered and mechanical aids must be relied upon.

The appliance used to cover a hard palate defect is known as an obturator and the section closing a soft palate defect an artificial velum. If there is an opening in the hard palate or if the pre-

maxilla is missing, the defect can be covered and the missing teeth supplied by an obturator. Obturators made of metal are far superior to those of vulcanite construction and produce less irritation. Some of the new light-weight alloys used in denture construction are excellent for this purpose.

For the soft-palate defect an artificial velum hinged to the posterior end of the obturator will follow the movement of the palate muscles and close off the nasopharynx. Soft rubber, formerly used for the artificial velum, disintegrates and becomes unclean.

Although surgery is the method of choice in correcting palate defects, a well-fitting prosthetic appliance serves a most useful purpose in cases where surgical relief is denied.

**Cooperation of the Orthodontist** — In the management of complete cleft lip and cleft palate the assistance of the orthodontist is essential in many cases after the surgery has been completed. Even when the alveolar border is well adjusted in unilateral complete cleft lip, the later development may not be sufficient to produce proper occlusion with the lower teeth. The central incisor on the cleft side may be rotated labially, the dental arch is often narrow in relation to the lower teeth, especially through the canine region, the lateral incisor and cuspid on the cleft side may erupt lingually in the sulcus of the repaired cleft, and the developing maxilla frequently fails to keep pace with the mandible, resulting in an increasing prognathism. These problems can usually be solved by the orthodontist. In bilateral cleft lip there is generally displacement of the premaxilla and even after careful replacement the premaxilla may not develop sufficiently to produce a normal anterior arch to support the lip and to carry the incisor teeth. The surgeon should be certain that all the nasal floor has been repaired. The orthodontist can then carry on his treatment to obtain a normal relation of the upper and lower arches so that the prosthodontist can replace any missing teeth.

**Cooperation of the Prosthodontist** — The aid of the prosthodontist may be necessary in unilateral complete cleft palate when the anterior teeth are so irregular and so displaced that the orthodontist is unable to align them in normal position. The arch can then be expanded, the useless teeth removed and a prosthetic appliance substituted. Prosthetic replacement is necessary in a large percentage of cases of bilateral cleft lip and cleft palate, due to an underdeveloped premaxilla which does not furnish support for the lip. The esthetic value of a well planned appliance which pushes the lip forward and corrects the profile cannot be overestimated.

In the event of previous operative failure, or when there is only enough tissue to provide a very short immobile soft palate, it is better not to operate as by so doing one may further complicate the construction of an obturator.

## CHAPTER XVII

### SURGERY OF THE FACE AND MOUTH

#### MACROSTOMIA AND MICROSTOMIA

THE mouth may be unusually large due to failure of normal coalescence of the maxillary and mandibular processes (macrostomia). One or both sides may be involved (Fig 125). In moderate forms, the edges of the cleft may be freshened and sutured. In extensive types, however, operation is seldom advisable.



FIG 125 —Macrostomia (Courtesy of Dr H S Vaughan)



FIG 126 Microstomia (Courtesy of Dr H S Vaughan)

Excessive fusion of the maxillary and mandibular processes produces an abnormally small oval aperture (microstomia) (Fig 126). The mouth may be enlarged by lateral incisions through the cheeks and suture of the skin and mucous membranes.

#### MACROGLOSSIA

The term is applied to a variety of conditions in which the tongue becomes so bulbous that it protrudes from the mouth.

In cases of lymphangioma, which are often congenital, the anterior portion of the tongue is more often involved than the entire organ. The enlargement may be progressive, remain stationary, or begin to enlarge at any period. (Refer to Lymphangioma.) In severe types the infant is unable to nurse and ulceration may

develop from the constant protrusion and dribbling. In such conditions a wedge-shaped portion may be excised from the central third of the organ or the tip may be amputated.

Macroglossia may also result from an abnormal increase of interstitial connective tissue. The condition occurs rather frequently in congenital mongolism and cretinism. In the latter type, thyroid therapy is frequently effective.

### CONGENITAL TONGUE-TIE

In congenital tongue-tie the tongue is bound to the floor of the mouth by an abnormally short frenum. Despite the popular belief that children who begin to talk late or develop speech defects are suffering from tongue-tie the actual condition is rare.

**Treatment.** Only cases in which the tip of the tongue turns downward when an attempt is made to protrude it should be subjected to frenotomy. The procedure is performed under light narcosis by slipping two fingers beneath the tongue, one on each side of the frenum, and snipping the edge with scissors, after which the separation is completed by blunt dissection and pushing the tongue backward. The blood vessels which run in the base of the frenum should be avoided. No postoperative care is required.

### ULCERS OF THE FRENUM

The ulceration usually results from abrasion of the frenum by the lower incisor teeth. The condition is commonly associated with protracted coughing, especially in pertussis. In cases of long standing a chronic inflammatory mass or granuloma may develop about the ulcer.

**Treatment.** The local application of a per cent silver nitrate solution is generally effective. Unusual cases which persist after subsidence of the coughing may require extraction of the lower incisor teeth.

### RANULA AND MUCOUS CYSTS

Although the term ranula has been loosely applied to any cyst occurring in the floor of the mouth, modern nomenclature restricts the appellation to cystic swellings which develop from obstruction of the salivary glands—parotid, submaxillary or sublingual. The more common tumefactions which result from obstruction of either a mucous or submucous gland or the anterior lingual glands of Blandin-Nuhn are classified as mucous cysts.

**Ranula.**—The causative obstruction may involve either the salivary duct or a main branch thereof. Although generally due to inflammation, a salivary calculus or foreign body is present in many cases. Apparently there is some etiologic factor other than

obstruction because complete calculus occlusion produces glandular sclerosis. Perhaps, as in hydronephrosis, intermittent or incomplete obstruction is the dominant element, as is illustrated by the following case. A colleague's child developed a sublingual ranula which persisted for two months. Saliva was aspirated daily and a filiform was insinuated into the duct on several occasions. The ranula persisted until a tooth-brush bristle was extruded.

**Symptomatology**—Ranulas may develop at any age and are occasionally present at birth. The parotid gland is seldom affected and most cases develop as a soft fluctuating cystic mass in the sulcus between the gum and tongue (submaxillary or submental origin). Growing slowly and painlessly, they may attain the size of a walnut and bulge in the floor of the mouth. Enlargement often occurs during mastication.

In most instances the excretory ducts are pervious to a fine probe or filiform and saliva or mucoid material is often obtainable by aspiration. Occasionally, a calculus may be palpated in the duct or "sounded" with a filiform. Although most calculi have a calcium base, a negative roentgenogram does not exclude their presence.

The majority of ranulas are unilocular and circumscribed, at times, however, large multilocular swellings diffusely invade the submaxillary soft parts and extend posteriorly to the base of the skull. They may be mistaken for simple cysts until their magnitude is discovered at operation.

**Acute Ranula**—In this rare type an excruciatingly painful cystic swelling develops suddenly in the floor of the mouth or in the submaxillary triangle. Increasing dyspnea may necessitate immediate surgery. The duct obstruction, due either to calculus or inflammation, is complete. Mild forms of acute ranula may be allergic manifestations. A case recently seen develops bilateral submaxillary swellings upon tasting vinegar. The attacks are painless and ephemeral.

**Diagnosis**—Ranula must be differentiated from mumps, mucous, thyroglossal and dermoid cysts, and cystic lymphangioma. Mumps is an acute self-limited infection, accompanied by pain and temperature and usually involves the parotid. Mucous cysts are small superficial circumscribed translucent swellings which appear just beneath the mucous membrane. The median position of lingual thyroglossal and dermoid cysts excludes them. Examination of the aspirated contents will distinguish ranula from cystic lymphangioma.

**Treatment**—Palliative treatment comprises repeated suctionage and the aseptic passage of a filiform or fine probe into the duct of Wharton, Stenson or Rivinus. The injection of escharotics is dangerous. Operative procedures comprise the oral removal of the salivary calculus, the insertion of one or two setons of heavy silk

from the lingual side, or excision of the cyst. The latter procedure is best performed through the floor of the mouth, the submandibular approach being rarely necessary. In extensive diffuse ranulas, the accessible portion should be excised and the remaining part swabbed with 95 per cent phenol, neutralized with alcohol, and the cavity packed with gauze. An extensive operation may be followed by glottic edema.

**Mucous Cysts**—Mucous cysts develop from obstruction of a mucous or submucous gland in the floor of the mouth, or rarely from the anterior lingual glands of Blandin Nuhn. They grow slowly and painlessly, appearing as a globular or ovoid translucent bluish tumefaction, just beneath the mucous membrane. They are usually unilocular and seldom exceed 2 cm in diameter. In rare instances, multilocular cysts attain large dimensions (Fig 127)



FIG 127 —Extensive multilocular cyst in the floor of the mouth. First noticed at six years.

**Treatment**—Aspiration, puncture or simple incision is almost invariably followed by recurrence. Excision of the lingual wall and cauterization of the base with 95 per cent phenol, neutralized with alcohol, generally results in cure. When unsuccessful, the thin sac should be excised. In extensive cysts, the lingual wall may be excised and the remaining portion cauterized with 95 per cent phenol, neutralized with alcohol, and the cavity packed with gauze.

#### MIXED TUMORS OF THE SALIVARY GLANDS

Mixed tumors of the salivary glands occur at all ages from infancy to advanced life, the majority being observed during the third and fourth decades. They develop most commonly in the parotid



gland infrequently in the submandibular and rarely in the sublingual. Similar growths are described as occurring in the mucous membrane of the pharynx and soft palate, cheek lip and lachrymal glands.

**Pathology**—Salivary gland tumors are pathologic puzzles and have caused much unsatisfactory speculation. Histologically they exhibit an extraordinary variety of epithelial and mesoblastic elements. A common type consists of tissue resembling *mucocartilage* arranged in lobules with a loose connective tissue framework. The cells, as in immature cartilage, rarely possess capsules and are often stellate. The nature of the cartilage is puzzling and many observers consider it to be of metaplastic derivation from the acinar glandular epithelium.



FIG 128—Mixed tumor of the parotid gland exhibiting growth after twelve years of latency.



FIG 129—Rapidly growing mixed tumor of the parotid gland. Fatal in seventeen months.

This definitely encapsulated type of tumor grows very slowly, often requiring ten or more years to attain a diameter of 1 to 3 cm (Fig 128). After remaining stationary in size for a number of years to all intent benign and quiescent, the tumor may suddenly grow vigorously and invade the parotid substance, neighboring structures, carotid vessels and lymph nodes. Death generally results from focal ulceration, sepsis, hemorrhage or metastases. The latter are usually pulmonary.

Another variety is composed of spindle cells interspersed with islands of *hyaline tissue*. The latter is prone to myxomatous degeneration and soft fluctuating areas may result therefrom. Mucin-filled cysts may also occur from secreting gland tissue which is often embodied in the growth. This type of tumor may grow steadily from its inception, have no quiescent period and prove fatal within twelve months (Fig 129).

**Symptomatology** Mixed tumors of the parotid usually begin as a small nodule in or upon the gland and develop within a capsule until of considerable size. The capsule may then gradually fuse with the gland substance. In some instances however the tumors are incorporated with the gland from their onset. The growths are generally firm in consistency cellular and mucoid types may be soft and even fluctuating. Multiple minute cysts are common and rarely the entire mass becomes cystic. Preponderance of one element may almost produce pure fibroma myxoma or chondroma. Malignant types are practically always epithelial basal-cell carcinoma myochoondrocarcinoma or adenoid cystic epithelioma. Sarcoma has been observed.

**Diagnosis** The slow growth and firm consistency of mixed tumors readily differentiates them from retention cysts due to salivary duct obstruction. (Refer to Ranula.) Rapid enlargement connotes malignancy.

**Treatment** Tumors of the salivary glands should be extirpated promptly. They are easily enucleated in the encapsulated stage and if the capsule is thoroughly removed with the growth recurrence seldom occurs. Malignant types rarely develop in children. They may be treated by radical surgery through sacrificing the parotid gland with the facial nerve or by radium therapy. Some malignancies are greatly benefited by the latter. A preliminary roentgenogram of the lungs should be taken in such cases to exclude metastases.

### ACUTE SUPPURATIVE SIALO ADENITIS

Acute suppurative inflammation of the salivary glands is uncommon. Although the condition may occur soon after birth as a sequel of obstetrical trauma most cases develop in children who are greatly debilitated or suffering from protracted illnesses such as typhoid fever or pneumonia. The salivary glands become involved either through hematogenous infection or ascending excretory duct invasion the usual organisms being the streptococcus staphylococcus or pneumococcus.

**Symptomatology** The patients are generally desperately ill. A chill may occur at the onset followed by pyrexia. The degree of the latter however is no index of the severity of infection. The involved gland most often the parotid swells rapidly and becomes acutely tender tense and hot. Its outline is often sharply demarcated and pus may exude from the excretory duct. Deglutition is painful and food is refused. Leukocytosis and polymucleosis are seldom high because of the child's low resistance. Bronchopneumonia is a common complication.

**Treatment** Breast fed infants should not be allowed to nurse as suckling may aspirate pus and contaminate the milk. When

bottle feedings are refused the normal body fluid balance should be maintained through gavage or cluses. For the latter 3 per cent glucose in physiologic saline solution may be administered subcutaneously or intravenously (Refer to Dehydration). The mouth should be kept clean. Local heat, infra red rays and short wave diathermy ameliorate the pain.

Although the inflammation may occasionally subside an area of fluctuation more commonly appears. Incision and drainage should be withheld until such softening occurs. In cases of parotid involvement the scalpel should be directed parallel to the fibers of the facial nerve and be carried no deeper than is necessary to obtain pus. A small cigarette or rubber dam drain is best employed. Hard tubing is dangerous. The prognosis is grave in debilitated infants.

### ACUTE RETROPHARYNGEAL ABSCESS

The retropharyngeal lymph nodes are situated on either side of the mid line in the loose areolar tissue between the pharyngeal wall and the prevertebral muscles. They vary from four to ten in early life but become fewer in number as the child grows older.

**Pathology** — The nodes become involved from the lymph drainage of neighboring or more remote infections of the nasopharynx, larynx, mouth, teeth or ears. The resulting adenitis may be hyperplastic or suppurative, the former eventuating in resolution and the latter in retropharyngeal abscess. The pus may rupture into the pharynx or burrow laterally and depending upon the integrity of the prevertebral fascia point in front of or behind the sterno cleido mastoid muscle. (Every case of cervical abscess should be examined for retropharyngeal abscess if the etiology is obscure.) In rare instances the process may extend into the posterior mediastinum or axilla. The carotid nodes especially the posterior chain are often involved secondarily.

**Symptomatology** — Acute retropharyngeal abscess is essentially a disease of early life and the majority of cases occur during the first year. The onset is generally insidious following a nasopharyngeal infection but in some cases there is no discoverable antecedent pathology. The abscess develops slowly during the course of several days and may attract no local attention until dyspnea or dysphagia appears. Fever is variable but persistent and the child's head is often held hyperextended to aid respiration. This posture in conjunction with the development of a nasal quality to the voice is very suggestive. Pharyngeal mucus is at times troublesome and nursings may be difficult and be regurgitated through the nostrils.

**Pharyngeal Examination** — When the abscess is located high pharyngeal examination may reveal a lateral bulging with over

The swelling occurs most often above and in front of the tonsil and the latter is displaced mesially. The surrounding edema is most marked in the uvula. The constitutional reaction is severe and the pain and dysphagia are associated with muffled phonation.

**Treatment** — The abscess should be evacuated as soon as it is demonstrable. To prevent possible damage to the deeper structures the scalpel should be covered with adhesive plaster except for its distal  $\frac{1}{2}$  inch. The incision is begun opposite the base of the uvula and extended downward in line with the anterior faucial pillar over the most prominent part of the abscess. An artery forceps may then be inserted to enlarge the opening. Aspiration of the pus should be avoided through suctionage.

## EPULIS

Epulis is a benign tumor which originates in the alveolar periosteum or periodontal membrane adjacent to a tooth or in the gap between teeth. The growth develops as a small smooth reddish or purplish mass which may be pedunculated or sessile and seldom attains large size.

**Varieties of Epulis** — There are two varieties. (1) Fibrous and (2) giant-cell epulis. The former is firm in consistency, grows slowly, is not prone to ulceration and corresponds to the periosteal fibroma of other bones. The giant-cell type is softer and often quite vascular, grows more rapidly and tends to ulceration and bleeding. Adjacent teeth occasionally become loose. Both types occur chiefly in children and young adults.

**Pathology** — The fibrous type is composed of strands of fibrous tissue similar in structure to ordinary hard fibroma. Such growths are definitely benign. The giant-cell type contains giant cells in a granulation tissue like stroma. At times the vascularity exhibits lymphangiectasis. The origin of the epulis variety of giant cell has occasioned much controversy. Some observers believe they are hypertrophied bone cells set free by absorption of the bone matrix, being identical with the osteoblasts of Kolliker. Others consider them modified endothelial or angioblastic cells. Mallory holds they are transformed wandering endothelial leukocytes. Although the tumors are essentially benign, recurrences follow incomplete removal and in unusual instances sarcomatous degeneration occurs. Sessile vascular types are potentially more dangerous.

**Treatment** — Except in small pedunculated growths the adjacent tooth or teeth should be extracted so that sufficient tissue may be removed to prevent recurrence. The epulis with its adjacent alveolus is then excised by a sharp rongeur. In vascular sessile types the alveolar removal should be more radical.

## ODONTOMAS

Primary tumors which arise from the osseous jaw are the same as those which occur in other bones and are necessarily of mesoblastic origin. Primary tumors that are not of osseous origin and which originate from embryonic tooth structures or misplaced remnants of tooth germs are termed odontomas.

The enamel body develops from an epithelial cord which grows inward from the primitive mucous membrane of the mouth whereas the papilla which forms the tooth pulp, cementin and dentin is of mesoblastic origin. During the development of the enamel organ and papilla a fibrous sheath or tooth sac surrounds the tooth germ. The tooth sac, enamel organ and papilla form the tooth follicle and tumors arising therefrom may thus be of epiblastic or mesoblastic genesis. The species comprises adamantinoma, fibrous odontoma, dentigerous cysts, root cysts and cementoma.

**Adamantinoma (Adamantine Epithelioma)** Adamantinomas are encapsulated growths derived from the enamoblast. Early stage tumors may be solid and contain branching columns of epithelium. Later from degenerative changes multiple irregular cysts develop which contain serosanguinous mucilaginous material and occasionally cholesterol crystals. In rare instances the growths become calcified.

**Pathology**—The tumors consist of islands of enamoblasts supported by a dense fibrous stroma which at times contains osseous particles. The outer surface of the islands is composed of cuboidal cells and the central area of fusiform cells or structureless material representing the stratum mucosum. Complex tumors may contain derivatives from the dentin, cementin or pulp structure.

**Symptomatology**—The tumors may develop in late childhood but are more common in young adults. They grow slowly and painlessly and swelling of the jaw is usually the only symptom. The tumefactions are smooth or slightly irregular and the overlying mucous membrane remains intact. Roentgenologically the growths appear definitely circumscribed exhibiting a mottled appearance when solid and an irregular cystic pattern after softening has occurred.

**Diagnosis** Sarcoma grows more rapidly and is destructively invasive. Epiulis is situated on the outer surface of the bone and exhibits no osseous involvement. Cystic adamantinoma may mimic a dentigerous cyst clinically but upon roentgen ray examination the latter contains a tooth within the cavity.

**Treatment**—Adamantinomas are benign tumors and do not metastasize nor invade the neighboring lymph nodes. Excision results in cure. Following the removal of a small growth the

cavity may be packed with iodoform gauze and permitted to heal slowly. Unusually extensive tumors may require resection of the jaw with subsequent autogenous grafting from the tibia or rib (It is advisable to remove the submaxillary salivary gland in resection of the lower jaw.)

**Fibrous Odontoma** — Fibrous odontoma consists of an unerupted tooth embedded in a thickened tooth sac. The tumors produce no symptoms and are accidentally discovered in routine roentgenograms. At times they resemble dentigerous cysts and the differential diagnosis may depend upon histologic examination. (Refer to Dentigerous Cysts.)

**Treatment** — Enucleation results in cure.

**Dentigerous Cyst** — A dentigerous cyst (follicular odontoma) contains the crown of a partially or completely formed unerupted and often misplaced tooth in a thickened fluid-distended follicle. The tumefaction is circumscribed and generally covered by a thin layer of bone within which there is a fibrous layer. The latter may be lined with granulation tissue or flat or cuboidal epithelium.

**Symptomatology** — Most cysts occur after the permanent teeth have erupted and develop slowly and painlessly as a superficial tumor in the outer side of the alveolar process. The osseous capsule may be so thin that a ping pong crackle is produced when it is pressed upon. The roentgenogram is characteristic: the cystic tumefaction is definitely circumscribed and contains the crown of a partially or completely formed tooth. Occasionally a follicular type of cyst developing deep within the bone is accidentally

diagnostic. Larger cysts may mimic the dentigerous type, absence of a contained tooth element excludes the latter.

**Treatment** — Small cysts at the roots of dead teeth usually adhere to the tooth when extracted. Large cysts may require the same treatment as dentigerous growths.

**Cementoma** — Cementomas (radicular odontoma) are of rare occurrence and arise from mesoblastic tissue of the papilla after the crown is formed. They are composed chiefly of cementin and develop slowly as painless circumscribed eburnated growths. Diagnosis is made from roentgen examination. Their density differentiates them from exostoses.

## PART V

# SURGERY OF THE NECK

### CHAPTER XVIII

### CYSTS AND FISTULÆ

#### THYROGLOSSAL CYSTS AND FISTULÆ

THYROGLOSSAL cysts and fistulæ are tubulo-dermoids which develop from the embryonic thyroglossal duct. In early fetal life the pharyngeal epithelium between the tuberculum and copula invaginates ventrally. Growing downward as a hollow epithelial stalk the lower extremity bifurcates to form the primordia of the lateral lobes of the thyroid gland. The remainder of the duct normally atrophies between the sixth and eighth weeks its original ostium being indicated by the foramen cecum.

When the duct fails to obliterate it traverses downward from the base of the tongue in the mid line of the neck to the hyoid bone and then along the anterior surface of the trachea to the thyroid isthmus. It usually passes through the hyoid but at times lies anterior or rarely posterior thereto. The portion above the hyoid is termed the *lingual duct* and that below the *thyroid duct*. Varieties of obliteration may produce cysts, fistulæ or accessory thyroid glands.

**Thyroglossal Cysts**—Thyroglossal cysts usually develop early in childhood occasionally they are present at birth and rarely the cyst formation occurs after puberty from inflammation in duct remnants previously quiescent. They occur as small cystic swellings which seldom exceed the size of a golf ball and may be situated above or below the hyoid bone.

**Suprahyoid Cysts (Lingual Dermoids)**—The tumefactions may develop at the site of the foramen cecum or occupy a lower central position in the tongue between the geniohyoglossi muscles. The latter type when large bulge in the submental space (fig 130). The cyst walls are composed of fibrous tissue lined with squamous epithelium and the contents consist of epithelial detritus, sebaceous material and rarely hair. At times the cysts are lined with ciliated epithelium. Bochradek found many fetal salivary gland acini



which open by lateral ducts into the lingual. The cysts containing ciliated epithelium probably originate from dilatation of these ducts (retention cysts).



FIG. 130 —Thyroglossal cyst filling the submental space and bulging in the floor of mouth. Present since early childhood.

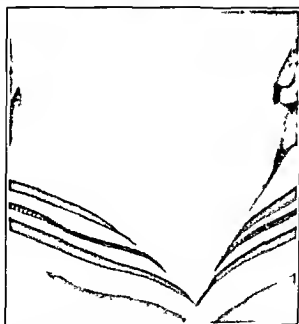


FIG. 131 —Infrahyoid thyroglossal cyst (slightly lateral)

**Infrahyoid Thyroglossal Cysts.**—These comprise the most common variety and occur in the mid-line of the neck (or slightly to one

side) between the hyoid bone and the thyroid isthmus (Fig 131) The cysts are relatively superficial and move up and down with deglutition They are lined with squamous or columnar epithelium and contain mucinous or sebaceous material and rarely hair Rudimentary thyroid tissue may be present in the walls

**Diagnosis**—A lingual cyst may be mistaken for an accessory thyroid gland which in rare instances develops between the geniohyoglossi muscles (Refer to Thyroid Gland) The aspiration of fluid is diagnostic Infrahyoid cysts must be differentiated from sebaceous cysts hygroma colli lipoma mid line superficial sequestration dermoids lymphadenitis and infrahyoid bursitis Sebaceous cysts are intradermal and are uncommon before puberty Hygromas occur as soft uni- or multilocular cysts generally in the anterior triangles of the neck and do not move with deglutition Lipomas are superficial lobulated tumors often connected with the skin and seldom occur in the mid line Sequestration dermoids are rare and develop as small cysts superficial to the deep cervical fascia Occasionally an enlarged submental lymph node is puzzling a focus of infection is usually demonstrable (Refer to Cervical Adenitis) In infrahyoid bursitis may develop from inflammation of the bursa between the body of the hyoid bone and the thyrohyoid membrane The swelling develops rapidly is acutely tender and seldom attains the size of a marble The condition may become chronic (Fig 132)

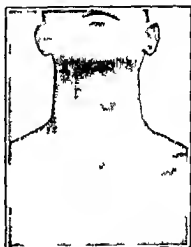


FIG 13 Bilateral Infrahyoid bursitis noted first when twelve years of age

**Treatment**—Cysts at the foramen cecum may be cured by excising the dome of the tumefaction and cauterizing the base with 90 per cent phenol neutralized with alcohol The deeper lingual types are best removed through a median submental incision Infrahyoid cysts frequently have a tubular process which extends upward a variable distance toward the base of the tongue In such instances the epithelial duct remnant must be completely excised with the cyst wall to prevent recurrence When the process passes through the hyoid bone the central 3 or 4 mm. of the latter should be excised with it Approximation of the divided hyoid is unnecessary

**Thyroglossal Fistulæ**—The vast majority of thyroglossal fistulæ are external and present a solitary opening at some point in the mid line of the neck between the hyoid bone and suprasternal

notch. The most common site is just below the cricoid cartilage and in some instances the ostium is slightly lateral. In rare cases of internal fistulæ the opening is at the foramen cecum and the sinus tract extends downward a variable distance between the geniohyoglossi muscles.

**Symptomatology**—Thyroglossal fistulæ are never congenital. They may develop soon after birth or even as late as puberty and result from inflammation and spontaneous rupture of preexisting cysts blind internal fistulæ or from the physician's meddling bistoury. The external ostium usually appears in a scarred depression and exudes mucus. The tract is generally palpable as a cord like process leading toward the hyoid with deglutition its opening puckers inward. In rare instances the sinus extends to the foramen cecum thus forming a complete fistula. The fistulæ are lined with columnar or at times ciliated epithelium and their walls may contain islets of thyroid tissue.

**Diagnosis**—The mid line location of the ostium with a cord like process extending toward the hyoid is pathognomonic. Rarely another type of mid line fistulæ is observed which results from faulty ectodermic fusion. The sinus tract in these cases is very superficial and may be attached by a fibrous cord to the mandible or the manubrium.

**Treatment**—Since the injection of escharotics is seldom successful complete excision of the fistulous tract is usually recommended. The procedure is best deferred until after the sixth year and is performed as follows. Through a vertical mid line incision after preliminary injection of the sinus with methylene blue the fistulous tract with a small amount of surrounding tissue is meticulously dissected upward. When the hyoid is traversed by the fistulæ its central 3 or 4 mm. is removed with the contained tract and the dissection is continued toward the base of the tongue to beyond the upper extent of the fistula. The soft parts are then approximated with No. 0 plain catgut the fasci with No. 0 chromic and the skin with fine dermal suture. Drainage is maintained for forty-eight hours. Whereas complete excision of the tract results in permanent cure incomplete removal is almost always followed by recurrence. Secondary operations through scar tissue are very difficult.

### BRANCHIAL (BRANCHIO-GENETIC) CYSTS

Branchial cysts are sequestration dermoid which result from faulty embryonic fusion. During the third and fourth weeks of fetal development five pharyngeal pouches appear on the lateral walls of the foregut. At corresponding levels the ectoderm indentates to form the branchial grooves or clefts. As the ectoderm

and entoderm approximate, the mesoderm is pressed aside and develops branchial arches between the clefts (Fig 7)

The first and second arches, the mandibular and hyoid, outgrow the others and form a gill like fold. The sulcus thus produced, termed the *cervical sinus*, is lined with epithelium derived from the ectoderm. During coalescence of the pharyngeal and cutaneous surfaces, islets of these epithelial cells may become sequestered. If they retain secretory function, a branchial cyst develops.

The majority of branchial cysts are lined with epidermis containing dermal glands, and their contents consist of sebaceous detritus and occasionally hair. Others are lined with squamous epithelium and contain mucoid material. The rare presence of ciliated epithelium indicates entodermic inclusion.

**Symptomatology** — Branchial cysts are rarely congenital and seldom occur before puberty, at which period there is an extraordinary development of many epithelial structures. They are more common in males, are of slow growth, and infrequently attain the size of an orange. Their usual situation is anterior to the sterno-cleido-mastoid,



FIG. 133 — Branchial cyst which developed at puberty

opposite the great cornu of the hyoid to which they may be attached (Fig 133). The cysts are deeply situated and when the smooth globular mass is pressed inward, a bulging may appear in the side of the pharynx.

**Diagnosis** — Branchio-genetic cysts must be differentiated from lymphadenitis, hygroma colli, thyroglossal cyst, cystadenoma of thyroid and adenoma of the parathyroid (Refer to sections thereon).

**Treatment** — Simple incision of the cyst should never be employed as a persistent sinus will result. Aspiration followed by the injection of escharotics is generally futile. The most satisfactory procedure is complete excision of the cyst with its lining membrane. The operation is one of considerable magnitude and requires intimate knowledge of the local anatomy.

## BRANCHIAL (BRANCHIO-GENETIC) FISTULÆ

Branchial fistulæ are tubulo-dermoids resulting from faulty embryonic fusion. The first or hyomandibular cleft is the primordium of the ear, auditory canal and Eustachian tube. With normal

development the other clefts coalesce and disappear. When such fusion between the sides of a cleft is lacking a branchial fistula results. The latter may be complete with openings in both the skin and mucous membrane or incomplete presenting only an internal or external ostium.

Depending upon their ecto- or entodermal origin branchial fistulæ may be lined with mucous membrane ciliated epithelium or skin. The mucoid discharge is often quite irritating.

**Symptomatology**—Branchial fistulæ are present at birth in contradistinction to thyroglossal fistulæ which are always acquired. In some cases the fistulous opening in the skin is so small that it escapes detection until the secretion attracts attention whereas in



FIG. 134.—B lateral branchial fistula of the first cleft (unusual)

others the ostium easily admits a probe. Occasionally a small tab of skin and cartilage (*cervical auricle*) surrounds the opening. The fistulæ are bilateral at times and there may be a familial history (Fig. 134).

**The External Ostium** The site of the external ostium depends upon the cleft involved. A line drawn from the external auditory meatus to a point just below the hyoid bone indicates the position of the first cleft from the anterior border of the sterno-cleido-mastoid at the angle of the jaw to the lesser cornu of the hyoid the second and from the same point to the suprasternal notch the third and fourth clefts (Fig. 135). The second cleft is involved most often.

The external opening may thus occur at any level along the

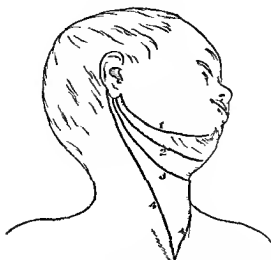


FIG. 135.—Locations of congenital fistulae arising in the first, second, third and fourth branchial clefts.

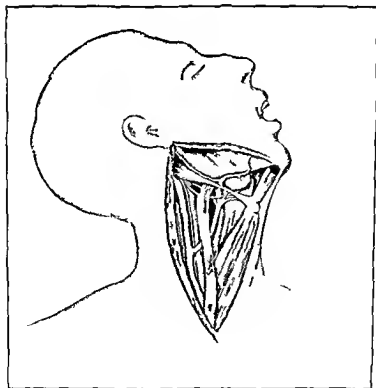


FIG. 136.—A branchio-genetic fistula and its relationship to neighboring structures.

anterior border of the sterno-cleido-mastoid. The most common sites are opposite the angle of the jaw, the upper border of the thyroid cartilage or just above the sternoclavicular articulation. The position of the internal ostium is constant in the lateral wall of the pharynx in or near the *sacculus pyriformis*.

**Complete Fistulæ**—The accompanying Fig. 136 indicates the course of a complete branchial fistula which opened externally at the suprasternal notch. The tract traversed upward along the anterior border of the sterno-cleido-mastoid lying upon the sterno-hyoid and sternothyroid muscles to the level of the cornu of the hyoid thence between the bifurcation of the carotid artery and beneath the posterior belly of the digastric muscle finally passing over the hypoglossal nerve to perforate the constrictor muscle at the site of the *sulcus pyriformis*.

**Incomplete Fistulæ**—External incomplete fistulæ should be examined roentgenologically after being injected with some radio opaque substance such as lipiodol. A deep tortuous tract is often demonstrated. Internal incomplete fistulæ usually remain unrecognized and asymptomatic unless they become inflamed when a pharyngeal abscess may result.

**Diagnosis**—A complete fistula is readily diagnosed by injecting methylene blue solution into the outer ostium, the dye soon appearing in the pharynx. An incomplete external fistula may be mistaken for a discharging sinus from a suppurating lymph node. The congenital history, absence of glandular swelling, mucous character of the discharge and the tortuous tract revealed by roentgen ray are pathognomonic. Thyroglossal fistulæ are never congenital, occur in the mid line and retract with deglutition.

**Treatment**—Because of the difficulty of extirpation attempts have been made to destroy the lining epithelium by the injection of escharotics. Although the results have been mainly disappointing, Cutler's solution has recently attracted attention and is worthy of trial. In case of failure, complete excision of the fistula should be performed.

**Operation**—Koenig's ingenious method for removal of the deep part of the fistulous tract is recommended. After the fistula has been dissected above the digastric muscle, one proceeds by blunt dissection to the region of the constrictor muscle. With the mouth held open, a probe threaded with silk is passed into the wound and made to bulge in the pharynx just anterior to the tonsil. A small incision is then made through the pharyngeal mucosa over the bulging point. The silk is tied to the fistulous tract and the latter is pulled into the pharynx as the probe is withdrawn through the mouth. After the free end of the tract has been amputated, the base is craterized and sutured into the pharyngeal wall. The soft parts of the neck are then approximated with No. 0 plain catgut.

the fascia with No. 0 chromic and the skin with fine dermal suture. Drainage is advisable for forty-eight hours.

Operative interference is best deferred until prepubescence. Unless the fistulous tract is completely excised recurrence will follow. Secondary operations tax the ingenuity of the most skillful.

### CERVICAL AURICLES

Small outgrowths composed of skin and cartilage termed cervical auricles are occasionally found along the border of the sternocleidomastoid muscle. Their removal may be indicated for cosmesis. An auricle may also occur at the external ostium of a branchial fistula (Fig. 134).

### CELLULITIS OF THE NECK

Cellulitis of the neck occurring in the areolar tissue superficial to the deep cervical fascia is of slight surgical import. With expectant treatment the infection either resolves or eventuates in a superficial abscess which responds to incision and drainage. Cellulitis occurring beneath the deep fascia however is a grave surgical pathology which is often lethal especially in debilitated infants.

**Etiology**—The condition occurs most commonly from septic throats in diphtheria and scarlet fever but may follow any invasion of the mucous membrane of the mouth or nasopharynx by a virulent organism predominantly the streptococcus. The inflammation generally develops in the submaxillary or submental regions and extends from the jaw toward the clavicle. At times the process is bilateral.

The following case typifies invasion following oral trauma. A boy aged twelve years had been treated some months for trench mouth. A bicuspid tooth was filled under novocaine anesthesia. The following day the submaxillary area became swollen and in forty-eight hours a brawny edematous tender mass extended to the clavicle. Temperature was  $105.2^{\circ}\text{F}$ , pulse 140, leukocytosis 17,800 and polynucleosis 91 per cent. Recovery followed prompt surgery. The wound cultures revealed hemolytic streptococcus.

**Pathology**—First described by Ludwig (1836) and often termed Ludwig's angina, the pathology of cellulitis of the neck is now recognized as a severe adenitis and periadenitis accompanied by bacterial invasion of the cellular tissues. The swollen and edematous lymph nodes incapable of coffer damming the infection form part of the confluent inflammatory mass. The process rarely subsides and usually eventuates in abscess formation or in extensive necrosis and purulent cellulitis which may extend into the superior mediastinum. Death may occur from sepsis, bronchopneumonia or rarely glottic edema.



**Diagnosis** —The acute development of a diffuse brawny edematous swelling without palpable nodes differentiates it from acute lymphadenitis. The condition is generally accompanied by hyperpyrexia, rapid pulse, prostration and at times delirium. Dyspnea and dysphagia develop frequently. Whereas high leukocytosis and polymorphonucleosis occur in sthenic types, a low count is exhibited in debilitated patients and in those overwhelmed by the infection. A chill may occur at the onset, repeated chills, however, usually indicate blood stream invasion. Blood cultures are sterile unless general sepsis develops.

**Treatment** —These cases can be grave concern. Prostrated children often refuse both nourishment and water and adequate maintenance of the body fluid balance becomes imperative. Glucose (3 per cent) in physiologic saline solution should be administered subcutaneously or intravenously in sufficient dosage to sustain such balance. (Refer to Dehydration and Acidosis.) Except in mild cases, immediate operation is definitely urgent. The incision is made over the point of maximum swelling, the scalpel being carried through the deep fascia. An artery forceps is then insinuated into the edematous mass and the blades are opened if pus is obtained. Failing to find pus after inserting the closed forceps in several directions, a soft rubber tube or cigarette drain should be introduced. The discharge in such cases usually becomes purulent within twenty-four to forty-eight hours. Virulent infections, however, may overwhelm the patient before this occurs. The presence of pus at operation indicates localization of the process and a favorable prognosis.

Antistreptococcus serum in the past has been disappointing. The concentrated serum of the New York State Department of Health has been employed at the New York Post Graduate Hospital with beneficial results in certain streptococcus pathologies. Recent reports concerning the administration of para-amino-benzene-sulfonamide have attracted favorable comment. Repeated blood transfusions are also of great value, especially when the convalescent progress is protracted.

## CHAPTER XIX

### CERVICAL ADENITIS

THE scalp face mouth nasopharynx accessory sinuses and intra are richly endowed with lymphatics which drain into various cervical lymph nodes. These may be divided into two groups (1) A circular chain (the pericervical) surrounding the base of the skull consisting of the parotid mastoid suboccipital submental and submaxillary glands and (2) a vertical chain comprising the retropharyngeal, the anterior cervical and the carotid or deep cervical. Fig 137 represents diagrammatically the course of the lymph flow from various parts of the head and face to the different nodes.

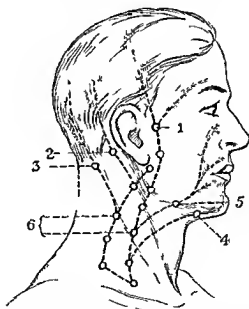


FIG 137 Lymphatic drainage to the various groups of lymph glands in the head and neck. 1 Parotid 2 mastoid 3 suboccipital 4 submental 5 submaxillary and 6 anterior and posterior carotid or deep cervical nodes.

The Parotid Nodes are three to six in number and lie superficial to the capsule of the parotid salivary gland, occasionally one or more is intracapsular<sup>1</sup>. Into this group drain the lymphatics of

<sup>1</sup> A temporary salivary fistula may follow incision of a deeply situated purulent node.

the forehead supra-orbital and frontal scalp regions the conjunctiva anterior surface of the pinna and the external auditory meatus. The writer recalls a child who had bilateral enlarged parotid nodes whose etiology caused much speculation. Treatment of chronic conjunctivitis resulted in subsidence of the adenitis. Fig 138 is that of a child referred to the clinic with the diagnosis of suppurative parotitis. The pathology proved to be suppurative parotid adenitis subsequent to a furuncle in the auditory canal.

**The Mastoid Nodes** receive drainage from the temporal region of the scalp and the posterior surface of the pinna and mastoid. Acute adenitis may suggest mastoiditis when the gland just below the mastoid tip is involved.

**The Suboccipital Nodes** comprise a chain of glands situated over the region of the suboccipital ridge. They receive lymph drainage from the occiput and are most often chronically enlarged from pediculosis capitis. The bites or scratchings cause excoriations whence infection is carried to the nodes.

**The Submental Nodes** two to four in number lie in close proximity to the sublingual salivary gland. Into them drain the lymphatics of the middle third of the lower lip the corresponding portion of the chin and the buccal mucous membrane covering the alveolar processes of the lower central incisors.

**The Submaxillary Nodes** four or more in number lie superficial to the submaxillary salivary gland occasionally one or more is intracapsular. They receive lymph drainage from the infraorbital and nasolabial regions the cheek upper lip outer third of the lower lip and the buccal mucous membrane covering the alveolar processes of all the teeth except the lower central incisors. The nodes are frequently enlarged due to the common incidence of alveolar infections in children.

**The Retropharyngeal Nodes** consist of four to ten glands which run vertically downward from the base of the skull on either side of the mid line in the loose areolar tissue between the pharyngeal wall and the prevertebral muscles. (Refer to Retropharyngeal Abscess.) Postpharyngeal peritonsillar and otitic infections drain into them. The glands seldom reveal their presence until a retropharyngeal abscess develops.

**The Anterior Cervical Nodes** consist of a few glands in the anterior region of the neck along the course of the anterior jugular veins. They receive lymphatic drainage from the neighboring skin and soft parts and are of slight surgical import.

**The Carotid or Deep Cervical Nodes** consist of an anterior and posterior chain which extend downward from the base of the skull anterior and posterior to the internal jugular vein and ultimately drain into the subclavian. The posterior belly of the omohyoid divides them into a superior and inferior group. The glands receive

extensive lymph drainage from the nasopharynx and also that from all the aforementioned nodes. They are most commonly involved from diseased tonsils and adenoids. The uppermost gland of the anterior chain, lying just behind the angle of the jaw and beneath the sterno-cleido-mastoid, is the first to enlarge and is often termed the tonsillar gland. With continued infection other glands become involved from above downward.

Knowledge of the tissue zones which drain into each group of glands is of distinct clinical value. Enlargement of the deep cervical glands suggests diseased tonsils and adenoids, submaxillary adenitis, dental caries, parotid adenitis, a furuncle of the auditory canal or conjunctivitis, and suboccipital adenitis a scalp nidus. It is seldom that the focus of infection cannot be demonstrated. Its eradication usually results in resolution of the adenopathy unless the nodes have suppurated, or are tuberculous.

Lymphatic tissue responds to irritation far more actively than any other tissue and this inflammatory hyperplasia is most marked in early life. Although the causative nidus is usually demonstrable, some cases develop without discoverable infection (idiopathic). Small palpable glands in the cervical, axillary and inguinal regions occur at times in healthy children and should not be considered pathologic.

Cervical adenitis occurs in the following schema of clinical conditions: (1) Simple hyperplasia, (2) Tuberculosis, (3) Hodgkin's disease and allied malignant lymphomatoses, and (4) Lues.

### ACUTE CERVICAL ADENITIS.

Acute cervical adenitis occurs often in infancy and early childhood as a complication of measles, scarlet fever and diphtheria. In older children, dental caries, tonsillitis and acute nasopharyngeal infections are common causes, the inciting organism being the streptococcus or, less often, the staphylococcus.

**Pathology**—The nodes exhibit active lymphoid hyperplasia, congestion, edema and not infrequently peradenitis. Depending upon the virulence of the infection and the host's resistance, the process may result in resolution or progress to suppuration. Severe infections may produce widespread cellulitis of the neck. The younger the child, the greater is the danger of suppuration and in debilitated infants resolution is uncommon. Occasionally the adenopathy remains stationary and the acute adenitis becomes chronic.

**Symptomatology**—The glands most commonly involved are those of the superior anterior carotid and submaxillary groups, subsequent respectively to tonsillitis or tooth infections. The adenopathy develops rapidly and in a few days several nodes may become involved. Occasionally a chill occurs at the onset. The nodes are

painful acutely tender and the overlying skin may be reddened and hot. Hyperpyrexia is usual and the patient may be prostrated. Suppuration commonly follows if the glands are larger than marbles and have matted from peradenitis (Fig 138). This generally occurs within ten days and seldom after the third week. In favorable cases resolution is often slow but is usually complete within ten weeks. Persistence of the adenopathy suggests tuberculosis. Acute cervical adenitis is rarely bilateral.



FIG 138.—Suppurative parotid adenitis secondary to furunculosis of the external auditory canal.

**Treatment**—When the adenopathy is secondary to tonsillar and adenoidal infection attention should be directed toward the nasopharynx: installations of a few drops of 2 per cent argyrol into the nostrils; topical applications to the tonsils; and the frequent use of warm antiseptic gargles. An ice-bag to the neck is comforting to older children with pyrexia; in infants and debilitated children warm applications are preferable.

Tonsillectomy should never be performed in the presence of acute adenitis. The procedure is also contraindicated when the glands

have subsided but the evening temperature remains elevated. The latter indicates active infection and the absorption from wide lymphatic areas laid bare through operation may precipitate fulminating cellulitis or provoke suppuration in a subsiding pathology.

In acute submaxillary adenitis secondary to alveolar infection prompt extraction of the offending tooth is advisable. A gum boil should be incised. Conservative temporization to save a tooth may result in suppuration of the glands.

**Suppurative Adenitis** No specific treatment will prevent suppuration. Various ointments such as ichthiol, guaiacol, iodine, etc., are useless and may irritate the skin. When suppuration develops incision and drainage should be deferred until an area of softening appears. A small transverse incision through the skin and fascia will permit the point of the scissors or artery forceps to open the abscess. The incision for submaxillary abscess should be at least a finger's breadth below the horizontal ramus of the jaw to avoid the branch of the seventh nerve supplying the levator anguli oris muscle. (Refer to Fig 142.) The facial vein should be preserved. A small soft rubber tube, rubber band, or paraffin gauze drain is employed for only a few days. Resolution is often slow and at times

usually under physical par and are classified as cases of lymphatism. They are non surgical, require hygienic dietetic treatment, and seem benefited by the administration of arsenic.

**Treatment**—Removal of the focus of chronic infection usually results in gradual resolution of the adenopathy. A proper hygienic dietetic regimen is also extremely important: wholesome food, abundant milk and eggs, fresh air, sunshine or ultra-violet irradiation, and adequate rest. Cod- or halibut liver oil or their concentrates and syrup of ferrous iodide are excellent adjuvants. The vitamin D potency of the fish oils is increased by the addition of viosterol.



FIG. 139 Bilateral adenitis of parotid lymph glands from chronic conjunctivitis

At the Home Hospital in New York City, the writer observed 92 children in whom there was definite enlargement of the cervical glands. All exhibited hypertrophied tonsils and adenoids. Following tonsillectomy and adenoidectomy the children attended an open-air school, had afternoon rest periods and slept at night on open-air porches. Whereas the weight gain for normal children of their ages was approximately 5 pounds per year, these children made an average gain of 10.6 pounds in nine months. The average time elapsed between operation and the disappearance of enlarged cervical glands was thirteen weeks. In 6 cases the nodes persisted and required roentgen therapy.

Ultra violet and roentgen ray therapy are of special value in obstinate cases which persist after the causative nidus is removed. Adenectomy is rarely required.

### TUBERCULOUS CERVICAL ADENITIS

This specific type of adenitis the scrofula of older writers results from infection of the cervical lymph nodes by either the bovine or human strain of tubercle bacillus<sup>1</sup>. The bovine type formerly so common and predominant in young children has been greatly lessened by veterinary supervision of cattle and pasteurization of milk. The human strain of the organism is generally transmitted through close contacts with parents or relatives who have open pulmonary lesions. Whether the bacillus is ingested or inhaled it lodges first in the mucous membranes of the mouth and pharynx, the usual primary nidus being the tonsillar and adenoidal tissues and less frequently the buccal mucous membrane covering the alveolar processes especially about carious teeth. There is no gross evidence of this invasion but careful bacteriologic studies have repeatedly demonstrated the organism's presence. The first clinical manifestation is enlargement of the cervical lymph nodes. This results from invasion of the glands by either the tubercle bacilli or their toxins following lymph drainage of the infected nasopharyngeal or oral mucous membranes.

**Glandular Involvement**—The anterior superior carotid lymph nodes are involved much more frequently than the submaxillary or parotid groups. (The anatomy of the lymphatics of the head and neck is described on page 253.) In practically all cases the disease is a focal process limited to the glands involved and secondary to a primary extralymphatic tuberculous nidus. The lymph nodes cofferdam the organisms and thereby tend to prevent blood stream invasion. Focalization of the disease is not always effective however. The coincident appearance of diseased glands in multiple areas unconnected by lymphatic channels as occasionally occurs in very young or debilitated children is definite evidence of a hematogenous source of infection. Also in rare instances the lymph nodes may become invaded through the blood stream when there is tuberculosis elsewhere in the body.

**Pathology**—It is seldom that only a single lymph node is diseased. Several glands are usually affected and at times the entire chain becomes involved. The first cofferdamming node is the largest, the others diminishing in size from above downward as they become progressively infected. Although the adenopathy results from

<sup>1</sup> Very rarely a case caused by the avian strain is reported. Such cases probably result from eating infected eggs.

invasion of the glands by the tubercle bacilli or their toxins low grade pyogenic organisms may produce a mixed infection

The diseased glands vary in size from that of a pea to a large olive With early involvement the gland surface on gross section is pinkish gray and of uniform consistency but the trabeculae are only faintly outlined At a later stage multiple foci of necrosis appear Finally the entire glandular structure becomes destroyed and granular grayish detritus fills the capsule

Glands which have enlarged to the size of marbles almost always contain necrotic foci

The findings are typical of tuberculous inflammation proliferation of epithelioid and giant cells in a surrounding zone of round cell infiltration Cheesy degeneration may occur in the center of the tubercles and through coalescence of multiple foci the entire gland may resolve into an intracapsular abscess The pus remains sterile unless a mixed infection is superimposed

**Spread of Infection**—As the tuberculous inflammation approaches the gland capsule neighboring glands become matted from per adenitis With rupture of the gland capsule the pus burrows along the cellular paths of least mechanical resistance toward the surface The skin becomes livid and necrotic and the abscess ruptures spontaneously The discharge may continue for weeks or months and the sinus may close and reopen repeatedly or it may heal The ostium is undermined and presents a characteristic bluish red edge When healing occurs a puckered retracted scar results often with purplish discoloration

The above pathologic picture is that of the average case The adenitis may result in resolution fibrous encapsulation calcification or in suppuration The course of the disease is often one of exacerbations and remissions depending upon the patient's resistance Generalized dissemination is most common in very young children and their resistance increases with each succeeding year

**Hematology** The blood shows alterations in both the corpuscles and plasma Secondary anemia is quite constant In acute types there is slight leukocytosis and increase of the neutrophils In chronic cases leukocytosis is absent the lymphocytes and transitionals are increased and there may be slight eosinophilia Sedimentation rates are increased in proportion to the acuity of the process The platelets are little altered The intradermal injection of 0.005 to 0.07 mg. of Old Tuberculin produces a sharp skin reaction often with rise of temperature headache and malaise

**Incidence** Tuberculous adenitis occurs with greatest frequency between the ages of three and ten years being comparatively rare in infancy and uncommon after adolescence The incidence accords with the period of life when the nodes are most often irritated by the toxins of the exanthems and by absorption from alveolar and



and nasopharyngeal infections. It is also the age at which milk is the principal food. Whereas the disease was formerly very common in urban communities it now occurs only occasionally. Milk supervision, dental clinics, diphtheria and scarlet fever prophylaxis and the early segregation of adults with pulmonary tuberculosis have been dominant factors in controlling the source of infection.

**Clinical Types**—In acute diffuse miliary tuberculosis the cervical nodes may be involved as a result of the tuberculous septicemia. This is usually a lethal process in which the glands play a minor role. It is significant proof, however, that the lymph nodes filter blood as well as lymph. In infants and young children there is another rare type which apparently begins with invasion of the cervical glands and progresses to a generalized miliary tuberculosis due to ineffectual clogging of the organisms within the nodes. The usual clinical types occur either as an acute or chronic local adenopathy.

**Acute Tuberculous Cervical Adenitis**—These cases resemble closely the clinical picture of simple acute adenitis. (See page 200.) The glands enlarge rapidly but the local and constitutional reactions are generally milder than in pyogenic infections. Although moderate pain and tenderness occur frequently, hyperpyrexia and prostration are uncommon. Moreover a careful survey of the mouth and nasopharynx fails to exhibit any acute pathology which might be an etiologic factor. The usual finding is that of hypertrophied tonsils and adenoids without evidence of acute or recent inflammation. The adenitis may persist and in a few weeks progress to caseation necrosis or partially subside and terminate in the chronic form. Resolution is rare.

**Chronic Tuberculous Cervical Adenitis** The pathology develops chiefly in undernourished and underdeveloped children who have lived under insanitary conditions. Healthy appearing children, however, who have had every hygienic advantage are not immune. The glands enlarge insidiously so that it is often difficult to determine the date of onset. Although at first only one gland is involved it is exceptional not to find several when the patient is presented for examination (Fig 140). The groups most commonly involved in order of frequency are the superior anterior carotid, the submaxillary, the superior posterior carotid and the parotid.

The writer observed 30 cases of tuberculous cervical adenitis which occurred in children at the Home Hospital in New York City. In 23 cases (76.7 per cent) the swelling apparently began in the tonsillar gland, secondary to diseased tonsils and adenoids. In 7 cases both anterior and posterior superior carotid glands were involved. In no case, however, were the posterior nodes involved alone and their infection appeared to be secondary to that of the

anterior chain. The remaining 7 patients (23.3 per cent) presented adenopathies of the submaxillary nodes, dental caries with alveolar infections occurred in 6.

**Symptomatology** The adenopathy develops slowly and progressively, often with periods of remissions and exacerbations. The process is generally unilateral and there is a greater tendency for the nodes to become matted than occurs in simple chronic adenitis. There is little or no local tenderness and the constitutional reaction is mild or absent. (Approximately one-half of the aforementioned cases had slight evening fever.) The adenitis may exist for many months gradually involving additional nodes. Some cases remain quiescent for long periods and suggest a malignant lymphoma. Many proceed to caseous necrosis of one or more nodes with



FIG. 140.—Tuberculous cervical adenitis (bovine infection)

eventual pointing through the skin (Fig. 141). A small percentage undergo extensive fibrosis and produce hard nodules adherent to adjacent structures. Complete resolution seldom occurs in untreated cases.

**Diagnosis** The diagnosis is not always easily made. The acute type is often mistaken for acute hyperplastic adenitis until excision results or chronicity ensues. A positive tuberculin test is merely presumptive evidence; it denotes tuberculosis somewhere in the organism but not necessarily in the cervical nodes. A positive reaction in patients over five years of age has little value. The absence of a focus of acute infection and of marked local and constitutional symptoms favors tuberculosis. Acute adenitis of pyogenic origin usually follows in the wake of an acute primary process and is accompanied by pain, tenderness, hyperpyrexia and often

prostration. Simple acute adenitis occurs much more frequently than the tuberculous form.

Chronic tuberculous adenitis often requires study for diagnosis. The onset and course are insidious, and the glands tend to mat and caseate. There may or may not be a demonstrable nidus such as diseased tonsils and adenoids. Inquiry should be made as to the source of milk supply and of possible exposure to pulmonary tuberculosis. Is there a family history of tuberculosis? Has any close relative a chronic cough? Such factors, in conjunction with a positive tuberculin reaction are presumptive evidence of tuberculous infection. The general condition of the child may be good, more often, however, the patient is undernourished, fails to gain weight and may have slight evening fever. *Most glands which enlarge gradually and progressively over a period of months without demonstrable evidence of a source of chronic infection, or which persist after removal of such nidus are tuberculous.*

**Röntgenologic Examination**—Roentgenograms of the glands are negative unless calcification has occurred. Chest plates often reveal a wide hilus shadow from involvement of the tracheobronchial root nodes. It is well to remember that glandular tuberculosis affects chiefly children and adolescents and that the disease is uncommon in infants. When

*a positive diagnosis cannot be made biopsy of a gland is indicated.*

**Avian Tuberculosis**—In the very rare avian bacillary types, there is acute local pain and tenderness accompanied by hyperpyrexia. The diagnosis rests upon a positive reaction to Avian Tuberculin and a negative response to Old Tuberculin.

Tuberculosis must also be differentiated from the adenopathies occurring with chancre oris and the malignant lymphatoses such as Hodgkin's disease, pseudo hygroma cysticum colli, lymphatic leukemia, lymphocytic aleukemic leukemia lymphosarcoma or pseudo-leukemia, leukosarcoma, spindle-cell sarcoma of the lymph glands and endothelioma. (Refer to Malignant Lymphomatoses.)



FIG 141.—Cervical abscess secondary to tuberculous adenitis

**Treatment**—In the rare forms associated with diffuse miliary tuberculosis supportive treatment supplemented with repeated transfusions may prolong life. Occasionally an otherwise hopeless victim is salvaged. It is generally recognized that tuberculous adenitis is a local disease of the lymph nodes secondary to a primary extralymphatic focus of infection and that it occurs most commonly in patients of low resistance. Treatment therefore should be directed toward (1) measures which will enhance the patient's resistance (2) removal of the focus of infection and (3) local treatment of the adenopathy.

**Hygienic Dietetic Regimen.** Tuberculous children require ample rest, fresh air, sunshine and a high caloric diet with an abundance of milk and eggs. Cod or halibut liver oil or their concentrates rich in vitamin D are highly beneficial. Viosterol may also be administered to increase the vitamin efficiency. Hematinics are indicated for the secondary anemia and in severe cases repeated transfusions may be required.

**Value of Heliotherapy**—The admirable results obtained by Rollier at the Leysin Clinic are indisputable evidence of the curative power of heliotherapy in glandular and bone tuberculosis. The entire body is exposed to the sunshine and on cloudy days the ultra violet rays of sun lamps are substituted. The treatment is somewhat tedious and is not uniformly successful. High altitudes have no special advantage and many clinicians favor seaside sanatoria. Tuberculin treatment has lost favor.

**Eradication of Focal Infection.** Extralymphatic foci such as diseased tonsils and adenoids or carious teeth should be removed. If the anterior superior carotid nodes are smaller than marbles and exhibit no evidence of acute inflammation primary tonsillectomy and adenoidectomy is indicated. In many such cases the glands subside and in a few months resolution is complete. Persistence of the adenopathy is an indication for radiation, heliotherapy or surgery. Larger glands matted from peradenitis almost always contain necrotic foci. In these types primary adenectomy is the safer procedure. The glands are badly damaged and if the tonsils and adenoids are removed first, acute suppurative adenitis or cellulitis of the neck may result. Tonsillectomy and adenoidectomy can be performed without danger during the postsurgical convalescence.

**Radiation Therapy.** In well selected cases radiation is of undoubted value. It is particularly effective in small glands without extensive peradenitis and in chronic sinus cases. One or two exposures to rays of high penetration will induce local inflammatory changes with subsequent regression if the radiation is to be effective. Additional exposures are useless and often harmful. In glands larger than marbles in which necrotic foci are usually present or in the presence of inflammatory changes evidenced by local tender

ness or evening fever, or in cases of extensive peritonsillitis, radiation is contraindicated. Radium therapy has few advocates.

**Surgical Therapy**—There is no unanimity of opinion as regards surgery. Whereas the ultraconservative would restrict surgery to the aspiration of an abscess, some opponents advocate wide excision of the glands as soon as the diagnosis of tuberculosis is made. Each case is deserving of careful evaluation, both from the physical and economic standpoint. In the majority of patients, rich and poor, surgery combined with heliotherapy is probably the most satisfactory treatment. Predicated upon the modern concept of the pathology, the disease is thereby eradicated.

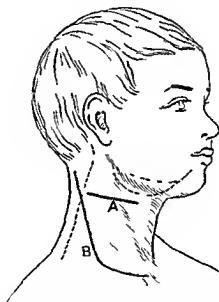


FIG. 142.—The dotted lines represent the course of the spinal accessory nerve and the variable course of the filers of the facial nerve to the levator oris muscle. Incision A is usually employed for the superior anterior carotid nodes and B when both the anterior and posterior chains are extensively involved.

**Operative Treatment**—Neck surgery in children should be performed under general anesthesia. Ethyl chloride and ether are the anesthetics of election. In older patients nitrous oxide, ethylene or cyclopropane with oxygen may be preferred. (Refer to chapter on Anesthesia.) All incisions, whether for palliative or radical surgery, should be made in the natural cleavage lines of the skin, as emphasized by Langer. These run transversely, or obliquely, inclining backward and upward. (See Fig. 142.) Such incisions tend to heal with hair-line scars where is vertical ones broaden.

Abscesses may be aspirated or incised and drained. The latter is the usual procedure. When a chronic sinus develops, radiation

will generally effect a cure with minimum scarring. In cases presenting multiple sinuses from spontaneous or incisional rupture, the pathology is usually extensive and associated with dense periadenitis. Unless acutely inflamed, radical adenectomy is the treatment of election. The skin openings, sinus tracts, diseased tissue and glands are all excised, the wound is then closed by careful approximation of the fascia, platysma and skin, except for drainage in the dependent angle. The mixed organisms in chronic sinus cases are usually so attenuated that primary union generally occurs. The author has repeatedly observed patients who have been treated for months by injections with various emulsions and eschareotic solutions. Many of these have been cured by operation within a fortnight.

**Operative Technic** —The usual types requiring radical adenectomy are those with multiple adenopathies of the superior anterior carotid or of the submaxillary nodes. In the former, a transverse incision in the natural cleavage lines of the skin of from 3 to 5 cm is usually sufficient. The incision, deepened through the platysma and fascia permits of wide retraction in all directions. Good exposure is imperative and one should never cut blindly. All pathologic nodes are carefully excised, preferably by scissor dissection, tissues which may contain veins should be clamped before being divided to prevent troublesome bleeding. Tissue soiling through the inadvertent opening of infected glands can usually be avoided by careful dissection. Injury to the spinal accessory, phrenic or hypoglossal nerve generally results from haste or inexperience.

**Submaxillary Adenectomy** —The transverse incision for submaxillary adenectomy should be at least a finger's breadth below the horizontal ramus of the jaw in order to avoid injury to the infra-maxillary branch of the cervico-facial nerve supplying the levator anguli oris (Fig 142). After the excision is completed, the fascia and platysma are carefully coapted with No. 0 plain catgut, and the skin edges approximated with horse hair or dermal subcuticular suture. Skin clips are preferred by some in older children. Careful closure of the fascia relieves tension upon the skin and aids in cosmesis. Drainage is required for a few days because of temporary lymphorrhea. Strands of silkworm gut or a small cigarette or elastic band drain, will suffice. A starch gauze bandage over the dressings to immobilize the head aids healing.

The operation of radical adenectomy, when skilfully performed produces little shock. Convalescence is usually rapid and the end-results are highly satisfactory. Cases with extensive glandular involvement may require a second incision, or the procedure may be performed in two stages. In certain instances partial adenectomy, followed by radiation, may be elected.

## LUETIC CERVICAL ADENITIS

Involvement of the lymphatic tissues of the body is a constant manifestation of early syphilis and the focal tell tale bubo accompanying chancre oris is well recognized. The negro is especially susceptible to lymphoid hyperplasia and in both early and late syphilis the lymph nodes occasionally reach such dimensions as to suggest leukemia.

Confusing examples of diagnostic difficulty may arise in differentiating gummatous from tuberculous cervical adenitis. The former is definitely rare except in negroes. Both conditions may pursue an identical clinical course with peradenitis matting softening and sinus formation. A Wassermann test should always be taken and if positive antiluetic treatment should be employed for at least two weeks before surgery or irradiation is begun. This therapeutic test is invaluable as gummatous adenitis subsides rapidly under arsphenamine. Biopsy is unsatisfactory as the pathologist is often unable to distinguish gummatous from tuberculous adenitis.

## CHAPTER XX

### THE THYROID AND THYMUS GLANDS

#### THE THYROID GLAND

SURGICAL disease of the thyroid gland is comparatively rare in infancy and childhood. The pathologies which concern the pediatric surgeon are simple or endemic goiter, hyperthyroidism and exophthalmic goiter, tumors of the gland and thyroiditis.

At birth the thyroid is relatively large and as the child develops it is subject to considerable variation in both size and conformity. Whereas the gland normally consists of two lateral lobes and a connecting isthmus, in approximately 40 per cent of cases a pyramidal lobe is present and in 10 per cent the isthmus is absent. At times the superior poles of the lateral lobes are lacking and in rare instances only one lobe occurs.

Aberrant thyroid tissue occasionally develops from lateral budding of the embryonic medium primordium<sup>1</sup> (thyroglossal duct). Accessory thyroids resulting therefrom may appear in the floor of the mouth above or below the mylohyoid muscles in the posterior triangles of the neck or in the suprasternal space. They are subject to goitrous enlargement and in such cases the normal thyroid may be atrophic or even absent.

#### ENDEMIC OR SIMPLE GOITER

Endemic or simple goiter is a diffuse hypertrophy and hyperplasia of all the elements of the thyroid gland, especially the epithelial. The parenchymatous process appears to be a compensatory physiologic response to a relative or absolute deficiency of iodine. The goiter *per se* is but a symptom of the disease whose specific etiology is unknown. Enlargement of the gland may normally occur at three pre-adult periods: the neonatal including the first few weeks of life, the prepubescent from eight to twelve years, and the pubescent from twelve to eighteen years. An exaggerated enlargement is termed goiter.

**Neonatal Goiter**—This form is rare and occurs most often in children born in goitrous districts or of goitrous parentage. Careful

<sup>1</sup>The ultimobranchial bodies were formerly thought to be late althymic in origin. There is no evidence that they are converted into thyroid tissue.



investigation usually reveals that the mother suffered a relative iodine deficiency during pregnancy. Most cases subside within a few months with or without iodine therapy. In rare instances a congenital goiter may surround the trachea and by pressure result in stillbirth (Fig 143). Cases have also been reported in which pressure symptoms have necessitated either division of the isthmus or partial thyroidectomy.

**Prepubescent Goiter**—This type is quite common, especially in goitrous belts. The compensatory hyperplasia and hypertrophy result from an iodine deficiency and the prophylactic use of iodine or its salts is accordingly valuable in potentially goitrous children. The insinuation that such therapy may produce hyperthyroidism is purely hypothetical. There is no scientific proof that iodine administration plays any part whatsoever in the genesis of hyperthyroidism.

**Pubescent Goiter**—This form occurs chiefly in girls and is analogous to the prepubescent type. The treatment is essentially medical. In some instances the goiter completely disappears and the gland returns to normal; whereas in others there is only partial subsidence and the thyroid remains somewhat permanently enlarged through colloid distention of the acini (*colloid goiter*). As involution proceeds, small nodules may develop which are often erroneously termed fetal adenomata (*nodular goiter*). The great majority are involutional nodules, the inconsequential end products of regressive and degenerative changes. In like manner, small cysts may develop from hyperinvolution (*cystic goiter*). None of these pathologies is a predisposing factor in the development of hyperthyroidism, nor does any play an essential role in the disease. Very rarely a hypothyroid state (myxedema) results from excessive involution.



FIG 143 Circular congenital goiter which apparently caused death from asphyxia. Thyroid at 6 enlarged.

## HYPERTHYROIDISM AND EXOPHTHALMIC GOITER

Hyperthyroidism is a sporadic idiopathic disease invariably associated with hypertrophy and hyperplasia of the epithelium of the gland and almost always with concomitant vacuolization of the colloid. The pathology may involve only part of a lobe, a whole lobe or the entire gland. The process begins most commonly in the right lobe and spreads through the isthmus to the left lobe.

**Symptomatology**—The disease is uncommon in childhood in infantile and even congenital cases have been reported. Most occur between the ages of fifteen and forty five years during which period the thyroid assumes its greatest functional activity. The four cardinal symptoms of hyperthyroidism are enlargement of the thyroid, tachycardia, tremor and elevation of the basal metabolic rate. Although only one or more of the foregoing may develop, elevation of the basal metabolic rate is constantly present. Exophthalmos may or may not occur and usually denotes severe thyrotoxicosis.

Instability of the autonomic nervous system is often a familial trait in pubescent hyperthyroidism. An antecedent history of tonsillitis, rheumatism or scarlet fever is also rather common. The onset may be insidious or precipitate. In some cases there is involvement of the thymico-lymphatic system manifested by enlargement of the tonsils, cervical and axillary lymph nodes, spleen and thymus gland. The differential blood formula may exhibit a relative lymphocytosis of 30 to 60 per cent.

**Treatment**—Exhaustive medical treatment including radiotherapy is always indicated in young patients before considering surgery. They are extremely sensitive to every form of stimulus, are poor operative risks and require meticulous handling. When all palliative measures fail, operation should be performed during a regressive phase if possible. Preliminary ligation and division of one superior thyroid artery followed some days later by a similar procedure on the opposite side is probably the safest operation in children. Partial thyroidectomy may be performed later if necessary.

The choice of anesthetic is important. Local infiltration is contraindicated due to the ill-effects of fright. One hour and a half before operation a child of twelve to sixteen years should be given  $\frac{1}{2}$  to  $\frac{1}{4}$  grain of morphine sulphate and one hour later tribromethanol (vertin) should be administered rectally (80 mg. per kilo weight). Cyclopropane, ethylene or nitrous oxide diluted with oxygen throughout the operation are the safest anesthetics. Toxic goiter with its increased oxygen metabolism and increased pulse rate calls for an anesthetic which is efficient in high oxygen dilutions. Cyclopropane is probably the best agent as it is still anesthetic in 80 per cent of oxygen. (Refer to chapter on Anesthesia.) Fortunately most all cases of toxic goiter in children respond to medical treatment and the surgeon is seldom consulted.

## THYROIDITIS

Acute thyroiditis is very uncommon in early life and the chronic type is exceedingly rare.

**Acute Thyroiditis** —The inflammation may involve part of a lobe, one lobe or the entire gland, and the process may be suppurative or non suppurative. The inciting organism is usually the streptococcus staphylococcus or colon bacillus. Most cases follow tracheitis apparently from retrograde lymphatic infection, puncture wounds and hematogenous infections are very unusual causes. At times epidemics of thyroiditis occur especially following measles.

**Symptomatology** —Most cases develop during an acute infection of the upper respiratory tract and the symptoms vary according to the severity of invasion. A chill often occurs at the onset followed by a sharp rise of temperature, extreme focal pain, dysphagia and inspiratory and expiratory stridor. The pain is paroxysmal in character and may radiate along the course of the second, third and fourth cervical nerves to the ears, face, arms and chest. The head is generally held still in flexion. Hoarseness from associated laryngitis is common and may progress to aphonia. Due to intra capsular tension the gland or portion involved becomes stony hard and acutely tender. With beginning suppuration the overlying parts become reddened and fluctuation replaces the hardened zone. Leukocytosis and polynucleosis are usually high. In rare instances tetanus may develop from extension of infection to the parathyroids.

**Treatment** —Mild cases subside in a few days and resolution may be aided by cold applications. Severe infections and those in which stony hardness is present demand immediate surgery. After exposure of the gland the capsule is divided and all focal suppurative areas are incised. A necrotic lobe is best excised. Hard zones require incision to relieve tension and prevent necrosis. Drainage should always be employed through the aid of soft pliable drains such as the gutta percha or cigarette type. Warm wet dressings afford relief and hasten resolution.

The prognosis is usually good in cases which receive timely and appropriate surgery. Delay in severe infections may result in fatal termination from edema of the underlying air passages. If a large portion of the gland has been destroyed, hypothyroidism may subsequently develop.

**Chronic Thyroiditis** —Chronic thyroiditis is exceedingly rare in childhood. The thyroid is relatively immune to tuberculosis and only a few cases of primary involvement are recorded. Even in miliary tuberculosis secondary invasion of the gland is uncommon. Enlargement may occur in early syphilis and gummata have been reported in congenital lues.

## TUMORS OF THE THYROID GLAND

Tumors of the thyroid gland are unusual in children and are practically always benign. Involutional residuals of previous hypertrophy and hyperplasia often termed colloid or fetal adenomas are not true tumors. (See page 269.) When these are excluded and they comprise approximately 90 per cent of the tumefactions the incidence of thyroid tumors is very small. The vast majority arise from the epithelium of the gland and are either adenoma or papilloma or an admixture of both. Fibroma and lipoma developing from the interacinar stroma are very rare.

**Adenoma.** This is the usual benign tumor of the thyroid and its genesis has awakened much controversy. Some observers insist the tumor arises from interacinar fetal remnants and that the epithelium resembles the cells of the fetal thyroid. They therefore term the growth fetal adenoma. Others claim the neoplasm arises from mature fully differentiated thyroid tissue that there is nothing in the morphologic character to suggest an embryonal origin and that the term fetal adenoma is a misnomer. There is also considerable debate concerning toxic adenoma. Certain patients whose thyroids harbor adenomas develop thyrotoxicosis. Whereas some would attribute the toxemia to the adenoma, recent investigation tends to exclude the neoplasm as an etiologic factor. It is becoming generally recognized that tumors of the thyroid like the various types of simple goiter neither predispose the patient to hyperthyroidism nor play an essential role in its development.

**Symptomatology.** Although adenomas of the thyroid may be congenital or develop at any age they are unusual in children under fourteen years. Occurring as circumscribed firmly encapsulated nodules they vary in size from grains to masses weighing several ounces. Multiple tumors may simulate nodular goiter and the antecedent history is important in differentiation. If the thyroid has previously been goitrous the nodules are probably involutional whereas tumefactions which develop in normal thyroid tissue are almost always adenomatous. Asymmetrical enlargement of the gland or the development of a solitary tumor also favors adenoma. The growth rate of the latter is usually slow and progressive. Rapid enlargement connotes hemorrhage or cystic degeneration.

**Papillomas.**—Tumors of this type are much less frequent than adenomas and present analogous characteristics. They are less prone to degenerative changes and often contain abundant colloid. Some types are an admixture of adenoma and papilloma.

**Treatment.**—Surgery is definitely contraindicated except in the presence of rapid growth or pressure symptoms. The integrity of the thyroid should always be preserved if possible until full maturity.

**Malignant Tumors**—These are exceedingly rare in children and but few cases have been reported. Rapidity of growth with fixation of the gland is a dominant characteristic. The great histologic lability and mobility of the thyroid renders a comprehensive classification of the malignancies impossible. Depending upon their origin and histologic growth the growths are grouped as carcinoma and sarcoma. Most cases develop in thyroids which have been subject to goitrous disorder for a long period of time.

## THE THYMUS GLAND

Experimental work upon the thymus gland has awakened much controversy. From the maze of conflicting evidence it may be assumed that under the influence of the thymus the other endocrine glands being in normal balance—the body takes on growth and accretion and secondary sex differentiation becomes inhibited. Upon this premise hyper- and hypothyroid types are accordingly predicated. The former results from subinvolution of the thymus with attendant overactivity or from a continuation of normal function beyond the time at which it should physiologically cease. The hypothyroid state follows precocious secretory cessation.

### THYMIC HYPERPLASIA

The surgeon's chief interest concerns thymic hyperplasia and especially its relation to sudden death from apparently trivial causes. The glandular enlargement may either occur independently or in association with other conditions such as status lymphaticus, hyperplasia of the tonsillar and adenoid tissues, rickets, exophthalmic goiter, leukemia and the malignant lymphomatoses. Its incidence in childhood according to various observers varies from 3 to 8 per cent. The vast majority of cases however are asymptomatic.

**Symptomatology**—Symptoms due to thymic hyperplasia occur most often in early life. The space between the manubrium and the spine in young infants is less than 2 cm. and glandular enlargement may accordingly produce compression of the trachea and bronchi with resultant respiratory symptoms.<sup>1</sup> These may be grouped as thymic stridor, thymic asthma and thymic death. In the first the chief symptoms are breath holding and both inspiratory and expiratory stridor. They may occur at birth or develop soon thereafter. In the asthmatic group the patients have recurrent sudden attacks of severe bronchial asthma. At times the latter may

The thymus normally increases in size to the age of two or three years and remains stationary until twelve. It then undergoes atrophy so that at full maturity only a trace or nothing remains.

follow the exacerbations of thymic stridor. In both types the dyspnea may cause grave cyanosis and collapse and even sudden death. The latter is apparently due to tracheal stenosis and laryngeal spasm.<sup>1</sup>

**Diagnosis**—It is in these conditions of status thymicus that trivial causes such as paracentesis, aspiration of an abscess or especially the induction of anesthesia may be suddenly lethal. Where there is the slightest suspicion of thymic hyperplasia roentgenograms should always be taken. The physical signs of thymic enlargement are notoriously unreliable.

**Treatment**—Elective surgery is definitely contraindicated in the presence of thymic enlargement. Radiotherapy should be employed in such cases until the hyperplasia is effectively reduced. Roentgen rays and radium appear equally efficacious. After thymic shrinkage has been induced operative procedures become relatively safe.

### HYPERTHYMIC TYPES IN OLDER CHILDREN

These children exhibit certain more or less definite characteristics attributable to the hyperplastic dysfunction. Such cases should be recognized for they are often surgically incompetent and operative procedures ordinarily fraught with minimum danger may lead to fatal outcomes. The children are prone to have a long thorax and trunk in comparison with the extremities and the thighs appear especially short. They may have soft skin with fine lanugo and seem younger than their age. The genitalia at puberty may not only be small but show lack of differentiation. The penis may emerge from a scrotal fold or the clitoris be large and pendant. Hair appears late and is sparse and in the male it assumes the female type of distribution. The joints may be so loosely bound that the arms and legs present a flail like appearance.

**Vagotonia and Adrenal Insufficiency** Hyperthymic patients often have low blood pressure, subnormal temperature and little endurance at times these vagotonic properties are marked. The vascular system may also exhibit small thin walled arteries. Especially important are the accompanying small and inefficient *valvules*. Under certain circumstances it appears probable that anesthesia, shock or emotion may produce collapse and death from rapid exhaustion of the adrenal reserve. Autopsies have also revealed hemorrhages into the adrenal, the ventricles of the brain and the myocardium. Tracheal pressure is seldom a factor in older children.

According to some observers thymic deaths are myths. They admit, however, that irradiation produces thymic shrinkage and symptoms at a relief.

### THYMIC HYPOPLASIA

Hypothymic states are thought to be produced by precocious involution of the thymus. Differentiation is too rapid and the old young type is produced. The epiphyses unite early, the stature is short and adult characteristics are prematurely superimposed. The permanent teeth erupt early and precocious puberty with secondary hair may develop even at the age of six years (Fig 144).

**Polyglandular Types**—In older children pure thymic types are uncommon. Whenever a grave deficiency exists an attempt at stabilization is brought about by compensatory measures. Thus to offset the vagotonic effects of the involuted thymus the adrenals, pituitary and thyroid may be called upon to perform such compensation through their sympatheticotonic properties. Superimposed characteristics may result therefrom and the types become modified accordingly.

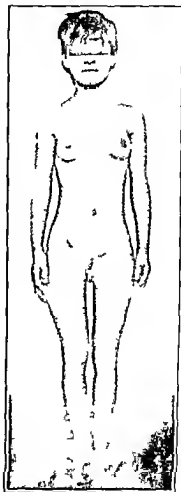


FIG. 144. Precocious puberty.  
Age six years and eight months.

### CYSTS AND TUMORS OF THE THYMUS GLAND

Cysts and benign tumors are rare. The latter comprise cystic lymphangioma, lipoma and congenital myxoma. The vast majority of thymic growths are malignant and are generally termed thymomas. Their structure varies considerably; some resemble Hodgkin's disease, others lymphosarcoma, while certain forms consist of round cells without reticulum cells.

**Symptomatology**—The tumors occur in the anterior mediastinum and usually surround and compress the trachea, bronchi, pericardium and great vessels. Dyspnea is generally the earliest symptom. Pleural effusion is also common. Most types grow aggressively and death results early from rapid cachexia.

## STATUS LYMPHATICUS

Cases of sudden death occurring in patients with thymic hyperplasia are grouped under the nomenclature of status thymicus. The same lethal phenomenon however may occur in other conditions as lymphoid hyperplasia and in hypoplasia of the heart, aorta and basilar cerebral arteries. Involvement of these different structures is variable. When lymphoid hyperplasia is the sole finding the condition is termed status lymphaticus. Although all the lymph nodes and the spleen may be involved the mesenteric and retroperitoneal glands are those most often affected. Hypertrophy of tonsillar and adenoid tissue is usually concomitant. In most cases of status lymphaticus however thymic hyperplasia is also present and the term status thymicolymphaticus best describes the condition.

These cases are most important from the surgeon's standpoint as sudden death may occur with the induction of anesthesia during the operation or within forty-eight hours thereafter. Unfortunately there is no positive criteria for diagnosing these patients preoperatively. A generalized adenopathy, enlarged thymus or inhibited sexual development should excite suspicion. Under such circumstances surgery should be interdicted unless it be imperative.



## PART VI

# THE THORAX.

By LOUIS R. DAVIDSON, M.D., F.A.C.S.

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### CHAPTER XVI

#### THE THORACIC CAGE

In the new born the antero-posterior and transverse diameters of the chest are approximately the same. With growth and development the transverse increases more rapidly and the chest assumes an elliptic shape.

#### ANOMALIES OF THE CHEST WALL

**Deficiency of the Pectoral Muscles** — This is probably the commonest congenital abnormality of the thoracic cage. The condition is always unilateral and may affect all the structures which normally occupy the pectoral region. The pectoralis major and minor muscles may be partially or completely absent and the overlying fat scanty. The mamma including the nipple may be small or absent and in 25 per cent of the cases the defect involves some of the ribs and cartilages including at times the adjacent margin of the sternum. The defect usually occurs between the second and fifth ribs and though variable in size and shape occupies an area which might be covered *in utero* by the child's fist or forearm. In approximately 1 case out of 7 there is an associated deformity of the hand on the same side, an error of dwarfing, syndactylism or club hand.

The pectoral defect interferes little with the patient's capacity for ordinary work unless the hand is also involved.

**Cleft Sternum** — In this condition the sternum is partially or wholly divided into two longitudinal bars by a medial fissure through failure of the parts to unite in the mid-ventral line. Ectopic cords may be associated with the anomaly.

In the simple type of cleft sternum the overlying skin may be normal as in a case reported by Love. The cleft extended as far as the fifth costal cartilage, the ends of the upper four cartilages on each side being connected by a fibrous or cartilaginous strip. During

may be pain and paresthesias beginning in the neck and radiating down the arm to the hand approximating the distribution of the ulnar nerve. The pain is often increased by exertion and cold. Secondary atrophy of the intrinsic hand muscles may follow. Occasionally there may be involvement of the sympathetic nervous system with resulting sweating coldness and dryness of the skin. Hoarseness may also occur from irritation of the recurrent laryngeal nerve. At times pulsation of the radial artery is greatly impaired.

**Treatment** — Asymptomatic cases require no treatment. If the child complains of pain and there is evidence of wasting of the small muscles of the hand operative removal of the cervical rib becomes indicated. The anterior transbrachial approach of Adson and Coffey is recommended. The fat and subareolar tissues in the supraclavicular triangle are reflected upward and laterally. The omohyoid muscle is divided and the dissection carried into the lower border of the posterior triangle. The transverse cervical and supra-scapular arteries having been ligated and cut the tendon of the anterior scalenus is sectioned at its attachment permitting it to be elevated medially. The cervical rib with its periosteum is then excised.

**Dorsal Ribs** — Infrequently an absence of one or more ribs or their fusion occurs. Concomitant deformities of the spine are associated.

**Cleido-cranio Dysostosis** — Cleido-cranio dysostosis affects only bones which are developed wholly or partially in membrane. The clavicular defect is usually associated with imperfect ossification of the cranial bones so that during infancy a large part of the vertex remains in its membranous condition the fontanelles being disproportionately large. When the skull changes are marked the bones of the face are often undeveloped and small producing a characteristic prognathism.

**Muscular Defects About the Shoulder** — The following are observed: absence of the clavicular portion of the trapezius; mal-development of the pectoralis major; absence of the clavicular portion of the deltoid; and when the clavicle is absent or represented by a small rudimentary bone the clavicular portion of the sterno-cleido-mastoid muscle is reduced to a thin band.

Fitzwilliams collected 60 cases of the following clavicular defects

	Left	Right
Both clavicles absent	6 cases	
One clavicle absent	9	0
One bone alone defective	1	3
Sternal end alone represented	23	27
Acromial end alone represented	1	2
Both portions present but united	14	17
Both portions joined but showing by angling notching or aching format on from two parts	5	9
Ligament prolonging inner ends outward	13	11

The defects seldom interfere with shoulder function or cause loss of power in the arm. No treatment is required except at times to support the shoulders from drooping forward.

**Acquired Deformities**—Rickets is responsible for most of the acquired deformities of the chest wall. Bending of the ribs at the costo-chondral junction is almost invariably the first appreciable bony change (rachitic rosary). Although usually small the nodules may be as large as marbles. In many cases there are lateral depressions over the lower third of the chest beginning at the ensiform cartilage and running downward and outward to the axilla corresponding to the attachment of the diaphragm (Harrison's grooves). The lower margin of the ribs often flares outward owing to enlargement of the liver and spleen and diaphragmatic pull. There is usually a marked diminution in the lateral and an increase in the antero-posterior diameters of the chest.

When any respiratory obstruction exists as hypertrophied tonsils or adenoids the abnormality becomes exaggerated. Irregular chest deformities may also depend upon the coexistence of certain pathologic conditions in the thoracic cavity such as atelectasis, pleurisy or pericarditis and spinal curvatures.

**Pigeon Breast (Pectus Carinatum)** The deformity is characterized by an increase in the antero-posterior diameters of the chest and a diminution of the lateral so that the sternum and costal cartilages are carried forward. The condition is generally associated with rickets particularly when respiratory obstruction coexists. Pectus carinatum may also occur with the kyphosis of dorsal Potts disease.

Treatment comprises (1) Cod liver oil or viosterol (2) ultra violet irradiation (3) thoracic exercises and (4) correction of any associated condition which causes respiratory obstruction as hypertrophied tonsils and adenoids.

**Funnel Chest (Pectus Excavatum Trichterbrest)**—The depression of the lower portion of the sternum carries with it the costo-chondral portions of the fourth to ninth ribs causing a marked decrease in the antero-posterior diameter of the chest at the xiphoid. Although a congenital type occurs the deformity is generally developmental. In extreme types the concavity of the sternum may so approximate the spine as to seriously compress the heart, great vessels and liver.

There are two schools of thought in respect to treatment. One advocates conservative measures regardless of symptoms while the other favors surgery when the depression compresses the heart and great vessels. Palliative treatment comprises a simple regimen of rest with postural and thoracic exercises.

Indwig Meir (1911) performed the first operation for funnel chest deformity in a patient with pulmonary tuberculosis. Two and a half centimeters of the second and third costal cartilages were resected and the patient was relieved of dyspnea. Sauerbruch in

his patient resected 3 cm of the fifth to ninth costal cartilages together with the left part of the sternum below the fourth cartilage. The patient was permanently relieved of both dyspnea and palpitation. In another instance Sauerbruch resected the fourth to the sixth left costal cartilages with the corresponding half of the sternum and filled the resulting defect with a fat graft from the thigh to protect the heart. This patient was also relieved of symptoms. Hoffmeister reports an operation by Lexer in which the fifth to ninth costal cartilages were divided and the corresponding portion of the sternum was removed leaving a gap of 15 cm between the divided ends of the cartilages on each side. Recovery was uneventful.

## INJURIES OF THE THORAX

Injuries of the thorax may be divided into two classes of wounds non penetrating and penetrating. The former will be discussed under (a) Contusions of the Chest Wall and (b) Lacerations of the Chest Wall.

**Contusions of the Chest Wall** — These may occur from a blow through pressure exerted against the chest in run-over accidents crushing between heavy objects or falls from a considerable height. The resulting injury may vary from a slight bruise to severe damage of the thoracic cage and its intrathoracic organs. Due to the elasticity of the ribs and sternum in young children crushing trauma may at times damage the thoracic viscera with only slight or absent external injury and without fracture of the ribs or sternum. The intrathoracic damage may involve not only the lung but the trachea, esophagus, great vessels or diaphragm. In many instances abdominal organs are injured at the same time especially the liver and spleen.

**Diagnosis** The first important factor is shock. This may occur immediately following the accident or be delayed an hour or more. In the latter instance shock is usually due to hemorrhage into the pleural cavity from laceration of the lung or tearing of the intercostal or internal mammary arteries. Hemoptysis may or may not occur. Hemorrhage into the pleural cavity exhibits the signs of pleural effusion and may be confirmed by aspiration. As the blood accumulates the pressure exerted by it on the torn lung may arrest the bleeding. If the mediastinum is freely movable the accumulating flow may cause serious pressure symptoms through pushing the mediastinal contents toward the contralateral side.

**Pneumothorax** may occur alone or in conjunction with hemothorax depending upon whether or not the underlying pulmonary tissue is lacerated. It may occur alone if the injury is limited to the trachea or a large bronchus. A serious type is the valvular pneumothorax which permits ingress of air into the pleural cavity.

but prevents its egress. As a result marked dyspnea and cyanosis develop from the accumulating air pushing the heart and mediastinum to the contralateral side and compressing the other lung.

*Mediastinal emphysema* arising from injury to the trachea or a main bronchus is a serious condition because of its pressure effect on important structures. Depending upon the degree of injury the air may involve not only the mediastinum but also the cervical structures and in extreme cases the subcutaneous tissues of most of the body. When the fractured end of a rib perforates the parietal pleura local or generalized subcutaneous emphysema may occur.

In laceration of the diaphragm abdominal organs may herniate into the pleural space. Symptoms may be absent and recognition be made only by roentgen ray. Hernia of the lung into the chest wall occurs infrequently following rib fractures.

**Prognosis** Injuries of the thorax associated with lesions of the thoracic viscera are accompanied by high mortality. Complications comprise empyema, pneumonia and fat embolism.

**Treatment** The most important factor is to combat shock. If due to hemorrhage immediate blood transfusion should be performed. If shock persists after the transfusion there is a possibility that the bleeding is still active and operation may be required to secure hemostasis.

In case of marked dyspnea and cyanosis aspiration of fluid or deflation of air should be resorted to until the patient is relieved of symptoms. Immediate laparotomy is indicated if there is evidence of injury to the abdominal organs.

Fractures of the ribs, sternum, clavicle or scapula are treated according to standard methods of therapy.

**Lacerations of the Chest Wall** These may be caused by projectiles, knives, glass, explosives, etc. It is important to determine whether or not injury has occurred to the underlying thoracic viscera. If there is no damage to the deeper structures treatment consists in controlling hemorrhage, removal of foreign bodies, debridement of devitalized tissue and the care of fractured ribs, sternum or clavicle. Tetanus antitoxin should be administered. The prognosis is usually good if there is no associated intrathoracic injury.

**Penetrating Injuries of the Chest**—Numerous organs may be involved by penetrating injuries and the pathology and symptoms vary according to the structures damaged. Shock is variable. Early symptoms may be caused by the accumulation of blood or air in the pleural cavity and later ones by infection. Marked dyspnea may be produced by a valvular type of pneumothorax or an accumulation of fluid which increases intrapleural pressure and presses the heart and mediastinal contents to the contralateral side with resulting compression of the other lung. Injury to the heart

and mediastinal emphysema likewise cause severe dyspnea. Injury to the pericardium and heart causes precordial pain, rapid pulse and increase in cardiac dullness. Hemoptysis may or may not occur. Mediastinal emphysema may be recognized by the accumulation of air in the subcutaneous tissues of the neck and the disappearance of normal cardiac dullness. Thoracic duct injury produces an accumulation of chylous fluid in the pleural cavity which may be confirmed by aspiration. When the diaphragm is pierced, symptoms and signs of intra-abdominal injury usually occur. Roentgen-ray of the chest following a penetrating injury is of aid in determining the presence of air or fluid, and the presence or absence of a metallic body.

**Complications**—Hemorrhage is the commonest complication. If the penetrating object pierces only the lung, the bleeding is usually slight, but if the heart, large vessels liver or spleen be injured, the loss of blood may rapidly prove lethal. Hemorrhage may also occur from injury to the intercostal or internal mammary arteries. The diagnosis of intrathoracic hemorrhage is made by the presence of shock, acute anemia and the signs of fluid in the pleural or pericardial cavities, confirmed by roentgen ray and the aspiration of blood. Intra abdominal hemorrhage may complicate the clinical picture.

Pneumonia frequently follows penetrating injuries of the lung because of the presence of bacteria in the bronchi and bronchioles. Empyema and lung abscess are infrequent complications. Pneumothorax is not serious unless it be of the valvular type.

**Treatment**—1 Combat shock with heat, large quantities of fluid blood transfusion and stimulants.

2 Débridement of wound with closure to prevent sucking.

3 In cases of marked dyspnea, aspiration of blood or deflation of air should be resorted to.

4 Penetrating injuries to the heart and pericardium require immediate operation. Hemorrhage from the intercostal or internal mammary arteries occasionally necessitates ligation.

5 Rifle or revolver bullets should only be removed if easily accessible.

6 Tetanus and gas bacillus antitoxin should be administered.

## INFLAMMATION OF THE THORACIC CAGE

Inflammation of the chest wall may be limited to the skin and subcutaneous tissues or involve the cartilaginous and osseous structures.

**Cellulitis**—Cellulitis of the subcutaneous tissues is caused by the entrance of microorganisms therein. Infection of the needle tract following aspiration in empyema is an occasional cause. Wet

tenderness in the affected bone. The skin over the swelling seldom exhibits the manifestations of acute inflammation and the leukocyte count is usually within normal limits.

**Diagnosis** — Diagnosis is based upon the history of typhoid infection, positive blood culture or Widal reaction and especially the growth of *B. typhosis* from the aspirated contents. The condition must be differentiated from syphilis, tuberculosis and sarcoma of the ribs.

**Treatment** — Treatment comprises vaccine therapy with or without surgical excision of the affected bone or cartilage.

**Tuberculosis of the Ribs, Sternum and Cartilages** — Tuberculosis of the ribs and sternum occurs frequently in children. The process may be secondary to contiguous pleural infection or begin in the medulla of the rib and gradually cause destruction of the cancellous structure with resultant bone abscess. Perforation of the cortex and the overlying thickened periosteum and fascia ultimately occurs and a soft tissue abscess is produced which may spread outward or laterally. If the advance is directly outward the overlying skin becomes involved and the resulting sinus formation leads to mixed infection. With lateral progression the cartilages and contiguous joints may become involved. The sternum is attacked in the same manner as the ribs and the changes occurring in it are similar. Perforation posteriorly is usually prevented by the firm layer of fascia which separates the sternum from the mediastinum.

Tuberculosis of the costal cartilage usually occurs from an extension of the process in the corresponding rib and infrequently from disease of the adjacent lymphatics. Two forms of onset are recognized: one starting in the perichondrium and the other in the cartilage proper. In the former perichondrial thickening develops with the formation of tuberculous granulation tissue and pus. Destruction of the cartilage follows and the associated swelling of the soft tissues presents the characteristics of a cold abscess. The process may remain localized with the formation of a sinus or spread to the corresponding rib and eventually involve other ribs. Dissemination is more frequent when the disease involves one of the lower ribs whose cartilages form a continuous bridge.

**Symptomatology** — The subjective symptoms are slight and because the disease is painless little attention is attracted to it until an abscess develops. The overlying skin seldom exhibits signs of acute inflammation. The swelling is soft and fluctuating with its long diameter in the direction of the rib.

**Diagnosis** — The insidious development of the swelling without the usual signs of acute inflammation and the aspiration of curdy pus is quite characteristic. The latter should be inoculated into a guinea pig and be cultured on special media to determine the presence of the tubercle bacilli. Roentgen ray of the chest will reveal bone changes in most instances.

**Treatment**—Rest heliotherapy good food and fresh air may suffice in the early stages. In general however surgical intervention is necessary. This comprises radical excision of the soft tissue abscess and the involved portion of the rib or sternum. When cartilage is involved Moschowitz advises its complete removal with partial resection of the bone at either end. Involvement of any rib below the fifth calls for subperichondrial resection of all the lower costal cartilages as there is a continuous bridge between them. After removal of the diseased tissue the wound is closed without drainage.

**Tuberculosis of the Sterno-clavicular Joint**—This rarely occurs in children. It may begin in either the clavicle or sternum or be primarily synovial.

The symptoms consist of pain swelling and tenderness. The swelling is soft and the overlying skin is not inflamed. When the clavicle is involved the tumefaction assumes an oblong shape.

Surgical excision of the involved area is indicated as soon as the diagnosis is made. The cavity is packed with iodoform gauze and the wound permitted to close by granulation.

**Syphilis of the Sternum and Ribs**—Gummata of the ribs and sternum may produce large fluctuating masses which at times eventuate in sinus formation. The condition is difficult to differentiate from tuberculosis without laboratory aid. The diagnosis is made by the presence of a positive Wassermann reaction absence of pulmonary tuberculosis as shown by roentgen ray and especially by the fact that the lesions heal under antiluetic therapy of arsenicals bismuth or mercury combined with potassium iodide.

**Actinomycosis of the Chest Wall and Ribs** Actinomycosis of the thoracic cage is usually secondary to involvement of the pleura and lung. The affected area becomes indurated of purplish red color and soon develops sinuses. The discharge contains minute friable yellowish or yellowish gray bodies which exhibit collections of the ray fungi. The latter may also be cultured from the pus.

**Treatment** Treatment comprises large oral doses of potassium iodide and curettement and cauterization of the lesion followed by roentgen ray therapy.

### TUMORS OF THE CHEST WALL

Tumors of the chest wall may be benign or malignant and involve (1) the skin and underlying soft tissues or (2) the osseous and cartilaginous structures.

**A Benign Tumors of the Soft Parts**—These comprise dermoid and sebaceous cysts angioma lymphangioma lipoma fibroma neurofibroma and melanoma. The respective pathologies are discussed in the chapter on Tumors of Childhood.

**Malignant Tumors of the Soft Parts**—Sarcoma arising from connective tissue and retaining most of the general characteristics



of its genesis is endowed with the power of invading and actively destroying adjacent structures and of forming colonies of its own tissue in distant organs. Rapidity of cell growth is a dominant feature and the tumor generally spreads so rapidly that there is insufficient time for the formation of encapsulating fibrosis. Treatment comprises surgical excision of the tumor, roentgen ray or radium therapy. The prognosis is generally grave.

**B Benign Tumors of the Ribs** — Pure *osteomas* are rare and most of the growths are an admixture of cartilaginous and osseous elements (*osteochondroma*). Both types are definitely benign and do not recur after removal.

*Chondromas* formed of hyaline or fibrocartilage tissue or both arise commonly in the cartilaginous portions of the ribs and at their junction with the sternum. The growths are definitely encapsulated, grow slowly and painlessly and form firm smooth or nodular tumefactions. At times they grow to an enormous size. (For further discussion refer to Chapter VIII.)

**Malignant Tumors of the Ribs** — *Ewing's sarcoma* of the ribs is usually found in patients approaching puberty. (Refer to Chapter XIV.) The tumors are quite rare and generally develop in the sixth, seventh or eighth ribs posteriorly. These locations are the sites of earliest ossification and accord with the view of Geschechter and Copeland that *Ewing's sarcoma* occurs in the skeletal parts where ossification begins toward the end of the second month of fetal life. Bergstrand reported 4 cases in young children in whom the growths extended into the pleural cavity by elevating the periosteum of the pleural surface of the ribs and pushing the pleura away. The process ceased at the attachment of the intercostal muscles.

*Osteogenic Sarcoma* — The pathology, symptomatology and treatment of osteogenic sarcoma is fully described in Chapter XIV.

**Malignant Tumors of the Clavicle** — In a series of 109 cases reported by Coley (1920) 30 occurred in children whose ages ranged from birth to eighteen years. The tumors were associated with recent trauma either in the form of a direct blow or severe muscular strain. Diagnosis is made from the history of pain, localized swelling of the clavicle, rapid growth, fairly characteristic roentgen ray findings and biopsy.

**Treatment** — Early total excision of the clavicle offers the only prospect of cure. The prognosis is generally unfavorable.

**Tumors of the Scapula** — Most tumors of the scapula are malignant. The benign growths are usually *chondromata* which in some instances undergo sarcomatous change. Treatment comprises early radical excision of the tumor.

In 65 cases of malignancy of the scapula reported by De Vancerede 20 occurred in children. Only one definite cure was observed following early amputation of the scapula.

## CHAPTER XXII

### THE MEDIASTINUM

#### EMPHYSEMA OF THE MEDIASTINUM

**Etiology** — Although mediastinal emphysema may occur with almost any pulmonary condition the most common factor is rupture of a pulmonary alveolus following violent respiratory efforts. The manifold causes comprise (1) Respiratory infections as pertussis, pneumonia, croup and influenza (2) injury to the trachea during bronchoscopy (3) injury to the lung during pneumothorax therapy or spontaneous pneumothorax. The free air may not only dissect its way interstitially and reach the areolar tissue at the root of the lung but may extend along the trachea to the floor of the mouth and in rare instances spread in the tissues over the entire body (4) rupture of a vessel as fracture of the larynx (5) following lobectomy.

**Symptomatology** The patients generally complain of discomfort or actual pain under the sternum and in the neck and also of crackling sounds on swallowing or moving the jaws. In many cases the symptoms disappear within thirty-six hours. In some instances, however, there may be pulmonary edema with labored respiration and cyanosis. Symptoms of compression of the axillary and femoral vessels may also occur. Roentgenologically the air is seen as an area of diminished density which separates the mediastinal pleura. When air encircles the heart the pleura appear as fine line shadows separated from the cardiac margins by the increased air.

**Treatment** Sedation may be required for the alleviation of pain or restlessness. If associated tension pneumothorax develops it should be relieved. (Refer to Pneumothorax.) Subcutaneous emphysema may occasionally require small multiple skin incision. In cases of extreme cyanosis venesection is indicated.

#### MEDIASTINITIS

Microorganisms may enter the area drained by the tracheo-bronchial nodes by the following routes: (1) Through inhalation into the lower respiratory tract. The organisms penetrate the epithelial lining and are carried to the tracheo-bronchial glands by the lymphatics and (2) by way of the blood stream.

**Classification** — Cases of mediastinitis may be classified as follows

1 Acute non suppurative mediastinitis

2 Acute suppurative mediastinitis

(a) Localized mediastinal abscess (These are rare in children and the majority occur in the anterior mediastinum)

(b) Diffuse phlegmonous mediastinitis

3 Chronic mediastinitis

**Acute Non suppurative Mediastinitis** The condition is usually accompanied by chills fever pain dyspnea dysphagia and hoarseness. It is frequently impossible to distinguish the pathology from suppurative mediastinitis without surgical intervention.

**Acute Suppurative Mediastinitis** The causative factors are numerous. (1) Perforation of the cervical or thoracic esophagus through swallowing of a sharp foreign body instrumentation or rupture of the organ following severe chest trauma. (2) Infections of the neck pus from a retropharyngeal or peritonsillar abscess may invade the mediastinum. The latter may also become infected from suppurative or non suppurative cervical adenitis acute pharyngitis or laryngitis. (3) Infections of the lung cases of acute mediastinitis have been reported in association with bronchopneumonia due to hemolytic streptococcus also with gangrenous inflammation of the lungs. (4) Exanthems mediastinal suppuration may occur during the acute exanthems of childhood or during the course of pyemia. (5) Bone lesions acute posterior mediastinitis may follow Pott's abscess of the spine and tuberculosis of the sternum may give rise to anterior mediastinitis. (6) Adenitis inflamed mediastinal glands may ulcerate into the esophagus.

**Pathology** — The commonest sources of suppuration of the mediastinum are cervical infections and traumatic perforation of the cervical or thoracic esophagus. Infections of the former may invade the mediastinum through lymphangitis cellulitis or direct downward extension from an abscess. The most frequent micro organism is the hemolytic streptococcus.

Mediastinal abscess may be single or multiple. The pus may rupture into the pleural cavity and cause empyema or erode the adjacent lung and perforate into a bronchus. Ulceration may also occur into the trachea esophagus or pericardium. When located in the posterior mediastinum the process may point in the supraclavicular fossa or extend from the posterior to the middle or anterior mediastinum. In rare instances the pus may burrow through the diaphragm and point in the iliac or femoral region.

**Symptomatology** At times the symptoms are extremely mild. Moderately severe and fulminating types generally parallel a septic course with chills fever and sweats. Pain in the thorax and beneath the sternum is a common symptom, with involvement of

the posterior mediastinum it is usually located between the shoulders or referred along the intercostal nerves. Dysphagia may result from swelling or pressure upon the esophagus and occurs most frequently in lesions of traumatic origin. Hoarseness, dyspnea, cyanosis and cardiac arrhythmia may be associated. Involvement of the vagus nerve may produce vomiting and that of the phrenic hiccough.

**Physical Examination**—*Physical findings* are wanting in many cases. In involvement of the anterior mediastinum with an abscess pointing anteriorly there is localized redness, swelling, tenderness and fluctuation. A large abscess in the superior mediastinum may produce dulness to flatness over most of the chest. In trauma to the cervical esophagus infiltration and tenderness of the organ are usually present. In some instances crepitations are noted.

The trachea may be displaced laterally or forward so that the finger cannot be inserted into the suprasternal notch. It may also become fixed and attempts to move it produce acute pain. Percussion tenderness is present over the spine in lesions of the posterior mediastinum.

A large abscess producing intrathoracic pressure may occasionally cause dilatation of the superficial cervical veins and in severe cases edema of the chest wall.

**Roentgen ray Findings**—The roentgenologic findings may be entirely negative, particularly if the lesion is situated near the diaphragm. With extensive involvement homogenous widening of the mediastinal shadow may be exhibited. In both localized and phlegmonous forms a focal widening occurs. An abscess may cast a globular shadow, a fluid level capping a shadow in the mediastinal zone is highly suspicious of mediastinal involvement, especially when the level crosses the mid line. It is of utmost importance to have films of the neck and chest taken in various positions.

**Diagnosis**—It is often impossible to make a positive diagnosis except at operation or autopsy. The importance of a careful history is self-evident when one remembers the relationship between mediastinitis and suppurative and traumatic cervical lesion. A history of trauma to the esophagus following instrumentation or the swallowing of a foreign body, retropharyngeal or peritonillar abscess or dysphagia accompanying a cervical abscess is of great aid toward establishing the diagnosis of mediastinitis. Physical examination, roentgen ray and bronchoscopic findings are important adjuvants. In cases secondary to disease of the sternum or vertebral column draining sinuses may be present. Their injection with lipiodol may aid in the diagnosis. Many cases of mediastinitis are erroneously diagnosed pneumonia.

**Prognosis**—In general the prognosis is bad. The inflammation is usually a complication of some serious disease rather than an independent infection. Cases with definite abscess formation which are recognized early generally recover if adequate drainage is established. Fulminating types with gangrenous phlegmon are rapidly lethal. The prognosis is influenced chiefly by the virulence of the infecting organism and the presence of complications.

**Complications**—Empyema may follow the rupture of an abscess into the pleural cavity or the pus may burrow through an intercostal space alongside the sternum and give rise to draining sinuses. In other cases ulceration occurs into the esophagus, trachea, bronchial tree or pericardium. In rare instances the process may burrow through the diaphragm and point in the iliac or femoral region. Suppurative pleuritis has also been observed complicating phlegmonous mediastinitis and may be bilateral.

**Indications for Operation**—When instrumentation or foreign body perforation is the etiologic factor immediate surgery is imperative. The esophagus should be exposed by an external approach and drainage instituted to the contaminated area. Operation should also be performed whenever a localized collection of pus is suspected. Whenever doubt exists as to whether the lesion is suppurative or non-suppurative surgical intervention is advisable.

**Surgical Procedures**—The mediastinal approach depends upon the location of the process. For periesophageal lesions an incision is made along the anterior border of the sterno-cleido-mastoid muscle. The carotid sheath is retracted laterally and the ribbon muscles and anterior belly of the omohyoid are severed if necessary. The lateral lobe of the thyroid gland is elevated and retracted medially, blood vessels crossing the field being ligated and divided. The dissection is then carried along the lateral wall of the esophagus. Good illumination is absolutely essential. The esophagus is lifted forward for entry into an abscess between it and the vertebral column and the abscess is laid open to its full extent. When extension into the mediastinum is suspected the lowermost part of the abscess must be visualized. The tract can be laid open through the cervical approach only as far caudad as the third or fourth dorsal vertebra. Drainage through the neck is inadequate below this level and a posterior mediastinotomy should be added when necessary.

The surgical approach to the posterior mediastinum depends upon the site of the lesion. Through a paravertebral incision a section of the selected rib adjacent to the transverse process is removed subperiosteally and part of the transverse process is excised. The parietal pleura is stripped away following which a rib with its transverse process above and below are removed. It is often impossible to enter the abscess without perforating the pleura. Should this occur the pleural cavity should be shut off by packing

or suture. The opening into the mediastinum must be extensive enough for thorough investigation.

**In low mediastinotomy.** Lilienthal uses an incision which starts over the ninth rib near the axillary line. It follows the rib toward the spine until the long spinous muscles are reached and is then continued upward parallel to the spine. The ninth or if necessary, the eighth and ninth ribs are resected. The pleura and periosteum are then pushed forward and as many additional ribs divided as are required for exposure. The high operation of Lilienthal is similar to the foregoing except that the sixth or seventh rib is resected first.

For exposure of the superior mediastinum the thoracoplastic flap of Kocher may be employed. The upper transverse incision follows the suprasternal notch and extends to the manubrium beyond both sternoclavicular joints. The sternoclavicular articulations are laid open and the sternal attachment of the left pectoralis major is severed. The incision is continued over the costosternal junctions of the first and second ribs. The lower transverse incision crosses the sternum at the level of both second ribs. The first and second right costal cartilages break like a hinge when the left border is retracted. The mediastinal structures are readily exposed by this approach.

The osteoplastic anterior mediastinotomy of Milton consists of a vertical incision from the base of the thyroid cartilage to the ensiform through the mid line of the neck and sternum. The sternum is divided and the ensiform cartilage is detached from the gladiolus. Through traction upon the divided edges of the sternum a gap of about 3 inches may be obtained. The wound is closed by uniting drill holes in the sternum with silk sutures.

The Sauerbruch operations known as anterior inferior horizontal and anterior superior longitudinal mediastinotomy are also widely used in operations upon the anterior mediastinum.

**Results of Operation.** The results obtained in cases of suppurative mediastinitis treated surgically are encouraging. Some surgeons report 66 per cent of operative recoveries. Cures under conservative forms of treatment are extremely rare.

**Chronic Mediastinitis.** Scar tissue formed during the healing of infections may involve important structures and seriously affect the individual. At times the fibrosis is secondary to involvement of the tracheo-bronchial lymph nodes from tuberculous, syphilitic or pyogenic infection. The symptoms depend upon the organs affected and their extent of involvement. Diagnosis and treatment are discussed in the section on Chronic Adhesive Pericarditis.

**Actinomycosis of the Mediastinum.**—The condition is secondary to actinomycotic lesions in the lung, neck or spine and occurs

most frequently in the posterior mediastinum. The abscess or abscesses may point through the thoracic cage and spread along the muscle sheaths.

## TUMORS OF THE MEDIASTINUM

**General Considerations** The symptoms produced by mediastinal tumors depend primarily upon the size, site and nature of the mass. Certain neoplasms may exist for a considerable time without producing any symptoms or findings. The more common manifestations are pain, cough, expectoration and dyspnea. The cough is often paroxysmal and hoarse or brassy. It may be unproductive or be accompanied by the expectoration of mucus and blood. Dyspnea, most marked on exertion, may be constant or paroxysmal. Expectoration is frequently an important finding. The presence of hair or sebaceous material is pathognomonic of a dermoid cyst or teratoma which has ruptured into a bronchus. Other symptoms due to compression of the tracheo-bronchial tree or the great vessels comprise cyanosis, dilatation of the superficial veins of the neck and edema of the face, neck and chest wall. Horner's syndrome may be found in ganglioneuroma and neurofibroma.

**Physical Examination**—This may elicit vocal fremitus over the tumor mass, dullness and absent breath sounds. Tumors of the anterior mediastinum may produce displacement of the heart.

**Roentgen ray Examination** This is often a valuable diagnostic aid and it is important to have films taken in various positions. Fluoroscopy may also yield information as to the size and location of the tumor. One of its values is in determining whether or not the tumor pulsates.

**Pneumothorax**—Artificial pneumothorax may aid in more clearly visualizing tumors which project into the pleural cavity from the posterior mediastinum. Biopsies may be taken from such growths by means of the thoracoscope. Bronchoscopic examination is valuable in excluding intrabronchial lesions or the encroachment of extrinsic masses upon the bronchi.

**Operative Treatment**—Various approaches to the mediastinum are discussed in the section on Suppurative Mediastinitis. Depending upon the size and location of the mass, an anterior or posterior approach is elected.

Tumors or cysts in the anterior mediastinum may be exposed through a T incision with the cross bar along the side of the sternum. Resection of the third and fourth costal cartilages and a portion of the ribs with division of the cartilages immediately above and below often gives adequate exposure. Approach through an intercostal incision, tripod or transverse sternotomy

may also be satisfactory. For cervical sternal dermoids a goiter incision combined with high median sternotomy is preferable.

**Benign Tumors of the Mediastinum**—Benign tumors comprise cysts fibroma neurofibroma ganglioneuroma lipoma chondroma osteoma and myxoma. They occur but rarely develop slowly and generally produce few symptoms.

**Dermoids and Teratomas**—This group includes a variety of neoplasms varying from simple dermoid cysts to complex teratoids termed by some investigators *fetus in fetu*. Simple dermoids are single or multilocular cysts lined with epithelium and containing sebaceous material hair and rarely teeth. The more complex teratoids may contain bone cartilage muscle thyroid gastro intestinal and nerve tissue. In a fetal parasite removed by Harrington the following tissues were found: skin hair and hair follicles sebaceous and sweat glands fatty connective lymphoid and nerve tissue body cavities lined by columnar epithelium with goblet cells and mucous glands suggesting the lower part of the gastro-intestinal tract masses of ossifying cartilage containing marrow smooth muscle and pancreatic tissue containing islands of Langerhans.

**Genesis of Dermoids** Teratoid tumors are considered to be congenital and various hypotheses have been advanced regarding their genesis. At first they were thought to be composed of tegumental structures that resulted from ectodermic inclusion at the time of closure of the primitive thoracic wall. Because they were believed to be derived from a single germ layer the ectoderm the term dermoid was applied. This is a misnomer as the tumors are usually of a more complex structure and contain tissues of both ectodermic and mesodermic genesis. Other theories were then advanced which favored various origins such as the thymus and thyroid glands the bronchial clefts and the bronchi.

These hypotheses were also inadequate as the presence of nerve tissue could not be explained thereby. The *bigeminal theory* of *fetus in fetu* was then evolved. Although many of the growths contain fetal tissues it is extremely rare for the tumor to be as highly specialized as the one reported by Harrington.

The tumors usually arise in the anterior mediastinum in front of the heart pericardium and great vessels. They may extend into the pleural cavities often become adherent to other structures and may ulcerate into adjacent viscera or through the chest wall.

**Symptomatology**—The symptoms are chiefly those of pressure substernal discomfort pain cough dyspnea and dysphagia. The pain is seldom focalized or severe but dyspnea may be extreme. Dilatation of the cervical veins and bulging of one side of the chest occur occasionally. There may also be mediastinal dullness and a fulness or boggy swelling above the sternum. Expectoration



of hair is pathognomonic of a dermoid which has ruptured into a bronchus

*Diagnosis*—The roentgen ray is of great value in diagnosis. A non pulsating spherical tumor projecting into the lung field may be exhibited in the anterior middle or superior mediastinum. The mass may be unilateral and sharply defined. Shadows of bone or teeth are rarely exhibited. Many of the tumors are quiescent and are accidentally discovered during examination or at autopsy (Figs 145 and 146.)

*Treatment*—Teratomas are potentially malignant and complete extirpation is accordingly the treatment of choice. Some surgeons

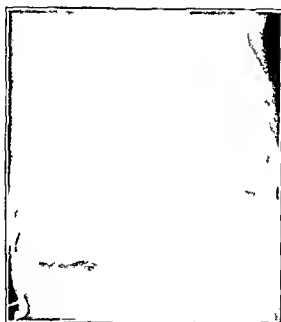


FIG 145 Teratoma of mediastinum

prefer to remove the growth in several stages while others elect a one-stage operation. An anterior or posterior approach may be employed. In the absence of infection or other complications the wound may be closed without drainage. Effusion often develops postoperatively and may require aspiration. Empyema necessitates drainage.

*Fibroma*—These rare tumors are often adherent to mediastinal structures, the pleura or diaphragm. They may remain asymptomatic indefinitely. The development of pain, cough, dyspnea and pressure symptoms depend largely upon the location of the tumor. Roentgen ray may reveal a clearly defined circumscribed shadow.

blending with the mediastinum. Tumors producing symptoms should be removed when possible.

**Neurofibromas** —The growths arise from the thoracic nerve sheaths, fibrous structures of the vertebral canal or the paravertebral sympathetic nerves and are usually located in the posterior mediastinum. The outstanding symptoms are those of pressure. Pain which tends to radiate along the intercostal nerves is an early and prominent symptom. Horner's syndrome may also appear. Treatment comprises surgical removal.



FIG. 146 —Teratoma bisected. Note cartilage and hair.

**Ganglioneuroma** —These rare tumors are of variable size and appear as elongated spindle or circular masses in the superior mediastinum. The mass may be unilateral or bilateral and casts a homogenous sharply circumscribed dense shadow. Treatment is excision.

**Lipoma** —The growths may be intrathoracic or extend from the thorax into the neck. The diagnosis is rarely made before operation or necropsy. Roentgen rays reveal a mass with a clearly demarcated outline, the center of which is often denser than the periphery. Lipomas producing symptoms should be removed when possible.

**Chondroma Chondromyxoma Chondromyxosarcoma** —These infrequent tumors generally arise from the costal cartilages and adjacent ribs and at times from the sternum or vertebral column. They are

circumscribed sharply demarcated and often nodular. The consistency depends upon the predominating structures. Although of slow growth pain is an early and common symptom. Cough and dyspnea usually appear later. With large tumors there may be evidence of mediastinal pressure suffusion of the face cyanosis and vascular engorgement hoarseness dysphagia and signs of pressure on the sympathetic chain. Roentgen rays may reveal a circumscribed nodular shadow. The tumors are subject to malignant changes and should be removed early.

### TUMORS OF THE LYMPH GLANDS

These comprise acute inflammation tuberculosis the malignant lymphomatoses including Hodgkin's disease leukemia and lymphosarcoma and metastases.

Normal hilar glands do not cast appreciable roentgen ray shadows. With increasing age the frequency of pulmonary and hilar infection increases and by the end of the first decade calcified areas and fibrotic changes in the hilar areas are commonly encountered.

**Acute Lymphadenopathy**—The condition is often demonstrable in generalized bronchopneumonia measles and pertussis. It may also be present in upper respiratory infections including sinusitis tonsillitis and nasopharyngitis. Diagnosis is based upon the history clinical course tuberculin reaction and roentgen ray findings. Paratracheal adenitis may be exhibited as a well-defined homogenous oval or globular roentgen shadow continuous with the mediastinum but not so dense.

**Chronic Lymphadenopathy**—Chronic enlargement of the mediastinal lymph nodes occurs in long standing pulmonary disease. It is found chiefly in asthma bronchiectasis lung abscess and chronic sinus disease. The roentgen ray shadow later takes on an irregular aspect due to fibrotic changes in the nodes. The outline of the mass is not clearly defined.

Tuberculous and luetic infections give rise to irregular glandular enlargement and the roentgenologic pattern may resemble lymphogranuloma lymphosarcoma or carcinoma. The diagnosis is determined by clinical and serologic findings.

**Lymphomas**—The term lymphoma has been applied by many investigators to include Hodgkin's disease lymphosarcoma leukemia and aleukemia. The tumors may be classified as follows the sclerosing type known as Hodgkin's disease or lymphogranuloma endothelial type of lymphoepithelioma lymphoplastic type or lymphosarcoma and the lymphocytic type (a) with leukemia and (b) without leukemia. (Hodgkin's disease is discussed in Chapter X.)

**Lymphosarcoma** — This malignant neoplasm arises in lymphatic tissue and has its own characteristic cell the lymphocyte. Its incidence is rare and females are more frequently affected than males.

Some observers believe the tumor has its genesis in thymic tissue, while others hold it originates from the mediastinal nodes. The small round-cell type predominates in early life and the growth usually occupies the thymic region and metastasizes freely. Death generally occurs early.

**Symptomatology** — The glandular enlargement is accompanied by fever, pallor, loss of weight and strength, and pressure symptoms: pain, cough, dyspnea and venous engorgement. Hemoptysis may also occur.

Roentgenologically, multiple rounded shadows appear surrounding the bronchi and great vessels. There is widening of the mediastinal shadow in the paratracheal, tracheobronchial and bifurcation regions. The enlargement is progressive and in the late stages the nodes become matted in a homogenous mass. Metastases to the pleura may be accompanied by pleural effusion.

**Diagnosis** — Lymphosarcoma responds more rapidly to deep roentgen-ray therapy than any other mediastinal tumor, and such response is a valuable diagnostic aid. The ultimate diagnosis rests upon biopsy.

**Treatment** — The tumor shadow may completely disappear after the first irradiation with roentgen rays. Recurrence of the adenopathy usually develops after a few months. The effect of treatment decreases with successive irradiations and the disease becomes rapidly fatal. Supportive blood transfusions may be helpful in the early stages.

**Leukemia and Aleukemia** — Some investigators believe there is a relationship between leukemia and lymphosarcoma and that if the life of an individual with lymphosarcoma is sufficiently prolonged the condition may change to lymphatic leukemia. Such cases have been reported following roentgen ray therapy. The relationship of the rays to the production of this change is unknown.

**Acute Leukemia** — Massive mediastinal growths in children are most commonly produced by leukemic tumors and the tumefactions may precede the typical blood changes. All leukemias, however, do not exhibit mediastinal adenopathy.

The tumors are usually soft in consistency and gray in color. They are composed largely of cells similar to those found in the blood stream in lymphatic leukemia. The regional lymph nodes are usually enlarged but remain distinct from the main tumor and the surrounding tissues are not invaded.

Tumors of round sarcoma-like cells found in certain patients dying from acute leukemia have been considered by many to be of thymic origin. The growths occur most frequently in the anterior

mediastinum and the term leukosarcoma has been applied to them by Sternberg

*Symptomatology* —The symptoms are chiefly those of pressure cough, dyspnea, cyanosis, edema of the face and dilatation of the cervical veins. Pleural effusion, usually on the left side, is not uncommon and there may be irregularity of the pupils.

*Diagnosis* —The tumors mimic lymphosarcoma and Hodgkin's disease and the diagnosis is made only by means of the blood picture. Tumors without blood changes cannot be differentiated from lymphosarcoma even upon microscopic examination.

*Treatment* —The effects of radiation are striking when employed before the blood changes of leukemia develop. Cases thus treated often show marked clinical improvement and respiratory embarrassment may disappear within twenty-four to forty-eight hours. Leukocytosis is also decreased but no marked change occurs in the differential count.

After blood changes develop irradiation is valueless. The duration of the disease is difficult to estimate as the mediastinal tumefaction may be present for some time before producing symptoms. The disease is generally fatal within seven months.

## CHAPTER XXIII

### THE PERICARDIUM.

#### ACUTE PERICARDITIS

PERICARDITIS occurring in children is always more serious than in adults. The condition is infrequently diagnosed and on that account is generally believed to be uncommon. Cabot reported 68 cases of purulent pericarditis in which only 7 were diagnosed during life and in Ponvton's group of 100 necropsies the clinical diagnosis was made only 6 times.

**Etiology**—Lobar pneumonia and empyema are the most frequent causes of pericarditis in early life and most cases occurring before the fifth year are due to these diseases. Thereafter, acute rheumatic fever is the commonest cause. Endocarditis which is frequently associated may or may not be accompanied by arthritis.

Infectious diseases such as scarlet fever, erysipelas, influenza or typhoid fever are occasional causes, also peritonsillar infections and osteomyelitis. Pericarditis associated with septicemia is often present in fatal cases.

Perforating wounds of the chest or ulceration of a foreign body through the esophagus into the pericardium occurs rarely. Disease of the ribs and sternum may occasionally spread to the pericardium. Tuberculosis is an infrequent cause.

An increase in the normal amount of pericardial fluid occurs in severe anemias and in general anasarca of cardiac or renal origin. Such effusions are seldom large enough to be recognized clinically.

**Pathology**—Either the visceral, parietal or both layers of the pericardium may be involved. In plastic pericarditis the membrane is injected and in severe cases is covered by a deposit of fibrin containing pus cells. Rheumatic pericarditis is usually of the serofibrinous type. The effusion containing flakes of fibrin and cellular elements may be either clear or turbid. Bloody fluid is extremely rare and is most often due to antecedent rheumatic fever (Ponvton).

Pneumococcus pericarditis is always acute and resembles pleuritis due to the same cause. In mild cases there is fibrinous exudate. With severer infection the pericardium becomes covered with a thick coating of fibrin and pus and the amount of exudate may reach a pint. The latter may be serofibrinous and become purulent or be purulent from the onset. (Most cases of purulent pericarditis in children are of pneumococcus origin.)

Tuberculous pericarditis is rare at any age and particularly so in early life. The infection may occur by way of the blood stream, the lymphatics or by direct extension. Most cases are secondary to tuberculous hilar adenitis. The process may present miliary tubercles upon the surface of the pericardium, pericardial adhesions or pericarditis with effusion. In the terminal stage there are extensive internal and external adhesions with caseous foci.

All varieties of acute pericarditis may eventually become chronic adhesive types.

**Symptomatology**—Pericarditis is often overlooked because its symptoms are frequently masked by those of the underlying etiology. Another reason is the condition is seldom considered.

A superficial friction rub is usually the earliest sign. It has a leathery quality, is synchronous with the cardiac cycle, to and fro in character and may be heard over the entire pericardium or only in discrete areas. In some instances it may be confined to a small zone at the base of the heart. The rub is not transmitted although it may be intense over the pericardium. Friction fremitus may also be present. With the accumulation of fluid the rub may be heard only over a restricted area at the base. It may disappear entirely or persist despite the presence of large effusion.

Pain and tenderness are inconstant findings. In rheumatic pericarditis pain is common. It may be referred to the abdomen and there may be upper quadrant resistance. It is seldom a feature in tuberculous pericarditis.

The symptoms in response to infection include circulatory disturbances, dyspnea and cyanosis out of proportion to the temperature and other evidences of toxemia. Pallor is often associated. The dyspnea may be accompanied by orthopnea and cardiac irregularity. It is the most outstanding symptom of tuberculous pericarditis and is out of all proportion to the lung changes present.

With the accumulation of exudate pressure symptoms arise and cardiac dullness extends progressively both to the left and right. With moderate effusions there is obliteration of the normal acute cardiohepatic angle (Roth's sign) and the area of cardiac dullness becomes triangular or pear shaped. With larger effusions there may be dullness from the right mammary line to the left axilla and the contour of the dullness may be almost circular. Widening of the parasternal dullness in the second interspace often occurs early. The dullness is shifting in character.

Dullness and flatness over the left side of the chest from the angle of the scapula downward is said to be due to compression of the left lung (Hamburger's sign). Bronchial breathing, bronchophony and egophony may occur over this area. In small effusions a zone of slight dullness may be present just inside the angle of the left scapula.

accompanied by faint bronchial breath sounds. Diminished tactile fremitus and fine rales occur just above. There may also be direct extension of the disease from the pericardium to the left lung. Antill states that massive collapse of the lung may occur from reflex disturbance of its nerve supply.

The apex impulse when palpable is not displaced and may be best elicited when the patient leans forward. With large effusions the heart sounds become weak, scarcely audible and displaced upward. At times the cardiac impulses are inaudible. The strength of the radial pulse is out of proportion to the apex impulse and paradoxical pulse is often present.

Fever usually occurs in the early stages of pericarditis. In purulent effusions it becomes remittent in character. Leukocytosis is commonly present. Variable pressure symptoms comprise dry reflex cough, dysphagia, aphonia, vomiting, engorgement of the jugular veins, enlargement of the liver and edema of the extremities. With progression of the pericarditis, frank heart failure may become manifest.

Tamponade of the heart is produced by large accumulations of fluid in the pericardial sac. The condition gives rise to the compression triad described by Beck: falling arterial pressure, rising venous pressure and a small, quiet heart. Beck states that all other manifestations are secondary to this triad. With the continuous increase of intrapericardial pressure a point is reached where symptoms of circulatory collapse appear.

**Roentgenologic Findings.** In the presence of pericardial effusion there is an abnormally shaped and enlarged heart shadow which changes with alteration of the patient's position. This finding is not encountered in any other condition. With moderate collections of fluid the heart shadow may be pear form and with large effusions pyramidal or bottle-shaped. There is also an increase in diameter of the supracardiac shadow and straightening of the left border of the heart. With massive effusions there may be associated collections of fluid in the pleural cavity of one or both lungs.

In poor fluoroscopic examination the cardiac pulsations are faint and in some cases absent. This finding is not of great aid as faint cardiac pulsation occurs in cardiac dilatation and may be due to myocardial damage.

**Electrocardiographic Tracing.** The changes in pericardial effusion are often similar to those found in the early stages of coronary closure. The alterations are of three types:

1. A decrease in the voltage of the *Q-R-S* complex. This is due to changes in the conducting medium surrounding the heart with consequent dampening of action currents.

2. Deviation of the *R-S-T* segment from the isoelectric level. This deviation usually occurs in the positive direction and results



from anoxemia of the cardiac muscle due to tamponage. The latter gives rise to compression of the auricles, hindering of venous return, lowering of the systemic and pulse pressures and interference with the flow of blood through the right and left coronary arteries. This involvement of the coronary arterial flow accounts for the positive deviation of the *R-S-T* sector in all leads.

3 Progressive changes in the *T* wave. These are associated with organization and repair of processes in the pericardium and in the underlying myocardium. There is usually inversion of the *T* wave, especially in Leads II and III.

The first two changes described are encountered during the acute phase of the disease. Alterations in the *T* wave generally occur after the acute episode has subsided.

In fibrinous pericarditis without fluid the changes in the *T* wave are not preceded by deviation of the *R S T* segment. Further proof that the *R S T* deviation is due to pressure exerted by the fluid is furnished by the fact that following inspiration these changes disappear. An increase in voltage of the *Q R S* complex is also frequently observed following withdrawal of the fluid. Inverted *T* waves noticed in the acute phase of pericarditis often disappear later.

**Diagnosis**—Pericarditis is diagnosed by knowing where to look for it. It should be constantly borne in mind that pneumonia in infants and rheumatic fever in older children are the dominant causes of pericarditis. In pneumonia typhoid fever scarlet fever and pneumonia, purulent pericarditis should be suspected with any sudden turn for the worse. Sudden sharp rise in temperature gradual disappearance of heart sounds previously of good quality increase in cardiac dullness, and sudden attacks of dyspnea and cyanosis strongly suggest the development of pericarditis.

In rheumatic pericarditis the close relationship between arthritis and pericarditis must be remembered. In some cases the arthritis occurs simultaneously with the pericardial involvement while in others it follows the first or subsequent attacks of pericarditis. The frequency of coexisting endocarditis in rheumatic cases is also important and it is necessary to differentiate pericardial friction rubs from endocardial murmurs. A history of chorea is seldom obtained in rheumatic pericarditis.

Tuberculous pericarditis is characterized by insidious onset fever, malaise, weakness, and loss of weight. The extreme rarity of pericarditis as the primary or sole manifestation of tuberculosis is of great importance in the diagnosis. Evidences of tuberculous involvement elsewhere should be found. The more important of these findings are enlarged hilar nodes or tuberculous infiltration of the pulmonary parenchyma.

The symptoms, physical signs and roentgenologic findings are

often of great aid in arriving at a diagnosis of pericarditis. In some instances, however, they are of little value. In the absence of acute fibrinous pericarditis the diagnosis is likely to be missed unless the effusion is large.

**Aspiration**—This is a valuable diagnostic aid especially in tuberculous and purulent forms. Examination of the fluid bacteriologically or by animal inoculation will reveal the causative organism. In purulent pericarditis aspiration should be used only for diagnostic and not therapeutic purposes.

**Prognosis**—This depends chiefly upon the type of the disease. Other influencing factors are early diagnosis and prompt treatment. In general the prognosis is unfavorable.

In rheumatic pericarditis both the immediate and remote prognosis is serious because of myocardial involvement. Lindley MacFarlane and Stevenson reviewed 51 cases of rheumatic pericarditis in children. Of these 24 died during the acute phase and 2 at a later period from cardiac decompensation.

Although suppurative pericarditis has been considered a very fatal disease, recent statistics afford a more hopeful point of view. Williamson reported 50 per cent of recoveries following operation and Winslow and Shipley 55 per cent. Early diagnosis and the institution of adequate drainage are imperatively essential. Pneumococcus pericarditis offers the most favorable prognosis and pyopericarditis due to staphylococcus or streptococcus complicating septicemia or acute osteomyelitis is the least favorable. Little is to be expected in tuberculous pericarditis as it is generally associated with advanced lesions elsewhere.

**Treatment of Pericarditis With Effusion**—Whereas conservative therapy was formerly universally recommended, surgical intervention is now widely practised in the treatment of certain types.

In rheumatic pericarditis many authorities believe that aspiration should be deferred since most rheumatic effusions subside spontaneously. They recommend the procedure only for the relief of distressing symptoms.

**Pericardiocentesis**—The aspiration should be carried out with the patient in a semirecumbent position in bed. Under local anesthesia a medium sized needle is introduced into the fifth or sixth left interspace close to the sternum. The needle should be slowly insinuated downward and outward until fluid is withdrawn. Other sites for paracentesis are through the left costoxiphoid angle at a point close to the ensiform cartilage opposite the seventh costal cartilage in the fifth or sixth interspace just within the outermost limit of cardiac dullness and in the fifth right interspace close to the sternal border.

In tuberculous pericarditis repeated aspiration and replacement with air is recommended. The pneumopericardium tends to retard

the re-accumulation of fluid and in some instances may prevent the formation of adhesions. It also lessens resistance for the heart to work against by substituting an easily compressible gas for a non compressible liquid.

**Treatment of Purulent Pericarditis** —Irrespective of the etiologic factor purulent pericarditis should be treated by early incision and drainage. Pericardiotomy with wide exposure should be the procedure of choice.

**Pericardiotomy** —Many methods of approach have been advanced and the choice of procedure is a matter of controversy. Simple intercostal drainage or the insertion of a trocar with continuous suction has been generally discarded as the drainage therefrom is inadequate.

Resection of one or more left costal cartilages is the method of choice with many operators. A portion of the sternum may also be resected. Graham recommends resection of the fifth sixth and at times the seventh left costal cartilages together with the lower angle of the sternum. The chondroplastic flap is turned upward in trap door manner. The internal mammary vessels are ligated above and below and the pleural reflection is pushed laterally exposing the pericardium.

The trans sternal approach of Malle is an excellent procedure in small children. The trephine opening made in the sternum should be large enough to permit exploration of the entire pericardial cavity with the finger in order to separate all adhesions.

In the xiphocostal approach of Larrey the cartilages of the sixth seventh and at times the fifth ribs are cut away the knife hugging the left border of the sternum closely. This exposure brings into view the left margin of the pleura and the internal mammary vessels both of which may be pushed to the left. The pericardium is then elevated with forceps and opened near the diaphragm. The exposure allows ample space to explore the pericardium.

A combination of this method with the trans sternal route has been used by Shipley. The trephine opening made in the sternum just above the junction of the gladiolus and ensiform is enlarged to the left until the lateral segment of the sternum and the ends of the fifth and sixth costal cartilages can be cut away. This exposes the triangle of safety or portion of the pericardium uncovered by pleura. The operator then has a bloodless field in which to work.

Allingham opens the pericardium from below by an incision through the diaphragm. The procedure obviates the resection of bone and cartilaginous structures.

Sauerbruch intentionally drained a pericardial effusion into the pleural cavity by making a communication between the pericardium and the left pleural cavity at the level of the fifth costal cartilage. The pleural effusion was treated by drainage.

Billings advocates the technic employed by Pool. The incision starting at the middle of the sternum at the level of the lower margin of the fourth costal cartilage curves downward to the upper margin of the left costochondral junction of the fifth rib and continues along the left costal margin to the middle of the seventh costal cartilage. It is then carried outward to follow the seventh rib. The soft parts are retracted and the seventh cartilage is divided at the sternum. The cartilage is then elevated and fractured 2 cm. from its sternal end and removed. The same procedure is followed with the fifth and sixth cartilages. The thin layer of tissue containing the internal intercostal muscles and posterior perichondrium is incised vertically and separated from the underlying parts. The *triangularis sterni* is then separated from the sternum and the underlying fat and edge of the pleura are displaced outward. The pericardium thus exposed is opened between forceps and the incision is extended downward to its reflection on the diaphragm. The edges of the pericardium are sutured to the skin or superficial parts to diminish the danger of mediastinitis.

Some surgeons recommend the insertion of a drainage tube around the right side of the heart and a second around the left and closure of the wound. Williamson is of the opinion that irrigation of the pericardium is of decided benefit and should be maintained until the fluid obtained is sterile.

**Postoperative Complications of Purulent Pericarditis**—The heart may temporarily stop beating following incision of the pericardium and in rare instances fail completely. It may be stimulated to re-beat by sharp slapping of the chest, lowering of the head of the patient, pulling the tongue forward, pressure on the pericardium with quick release or cardiac massage.

Secondary pocketing of pus in the pericardium occasionally occurs and requires relief. At times irrigation of the cavity with Dakin's solution is effective. In a small number of cases empyema thoracis develops.

### CHRONIC ADHESIVE PERICARDITIS

The condition is also known as mediastino-pericarditis and concretio pericardii. Pick's disease or pericarditic pseudocirrhosis of the liver is present in from 2 to 54 per cent of the cases coming to necropsy.

**Etiology**—Chronic adhesive pericarditis may result from any acute pericardial inflammation. Rheumatism is the chief etiologic factor and the pathology may follow a single or repeated attacks of rheumatic pericarditis. Tuberculosis is also an important cause. Of the pyogenic organisms the pneumococcus and streptococcus are encountered most frequently and the staphylococcus and

influenza bacillus but rarely. Cases due to the gonococcus have been reported by Huber and Bubis.

**Types of Chronic Pericarditis**—White groups chronic pericarditis into four types.

1. Adhesions which give rise to few or no clinical manifestations. These are usually between the pleura and the pericardium or between the pericardium and the heart.

2. Adhesions which give rise to an important degree of constrictive pericarditis without external adhesions.

3. Significant external adhesions to the chest wall and mediastinum (mediastino-pericarditis).

4. A combination of mediastino pericarditis and constrictive pericarditis.

The two most important groups are mediastino pericarditis and constrictive pericarditis.

In mediastino pericarditis the heart may be bound to the chest wall and systolic contraction be hindered thereby. Cardiac compensation may readily result from such abnormal fixation especially when myocardial damage coexists.

In constrictive pericarditis there may be extensive adhesions between the visceral and parietal layers of the pericardium. The two layers may be completely adherent and the heart lie encased in a thick inelastic shell (concretio pericardii). The heart becomes throttled; its chambers cannot expand in diastole and a marked degree of stasis, particularly of the caval system, results.

Any type of adhesive pericarditis may give rise to Pick's disease. The essential feature for its development is not the mere presence of adhesions but the compression effect exerted on the heart by the contraction of scar tissue. The main consideration is crippling of the heart. Obliteration of the pericardial cavity does not necessarily produce disability unless the pericardium is so contracted that expansion of the heart is interfered with.

**Symptomatology**—When the heart becomes exhausted through tugging on the chest wall or pulling upon the diaphragm the usual symptoms of congestive heart failure appear. Dyspnea is the earliest and most important; abdominal discomfort and distention may follow and general weakness is often pronounced.

In certain cases there may be a total absence of physical signs and the pathology may be accidentally discovered at autopsy. The usual symptoms comprise the following: cardiac enlargement due to hypertrophy and dilatation; fixation of the heart; hepatic engorgement and cirrhosis; marked ascites with little or no edema of the extremities; Broadbent's sign of systolic retraction with diastolic bulging; paradoxical pulse (Kussmaul's sign); sudden collapse of the veins of the neck with ventricular systole (Friedrich's sign); murmurs; auricular fibrillation in cases associated with Pick's

usually re-accumulates and the patient ultimately succumbs from cardiac decompensation or intercurrent infection. The sole hope for cure in advanced chronic adhesive pericarditis is surgery.

**Operative Procedures** — These are of two types (a) The Brauer operation or cardiolysis indicated in cases of external adhesions or mediastino pericarditis and (b) the Delorme operation employed in constrictive pericarditis to free the heart from its thick contracted shell of pericardium.

If adhesions occur only between the pericardium and thoracic wall removal of the pericardium is unnecessary. When the heart is surrounded by a leather like shell however rib removal alone is of no benefit.

**Cardiolysis (Brauer)** — This procedure has been performed in children with excellent results. The measurements of certain landmarks referred to are for adults and the mensuration for children will depend upon the patient's size.

The incision beginning over the third left rib 3 inches from the mid line is curved over the rib to the middle of the sternum. It is then extended downward to the junction of the mid line of the sternum with the seventh rib and continued over the latter to a point 2 inches from the sternum. The skin, fat and pectoralis major are dissected upward in one flap and reflected outward exposing the costal cartilages of the third, fourth, fifth and sixth ribs. The cartilages with 1 inch of the ribs are then removed leaving the posterior perichondrium. If necessary part of the sternum may be removed with rongeur forceps. If the pleura is opened by accident a purse-string suture is passed around the opening and the collapsed lung is reexpanded by positive intratracheal pressure. Air tight closure completes the operation.

Beck and Griswold advocate a horizontal skin incision through the center of the sternum with two cross arms at each end. The effect produced resembles the letter H. The cartilages can then be removed from both sides.

If the rib segments regenerate the resistance offered by the thoracic wall will again develop. Various chemicals such as Zenker's fluid have been used to prevent osteogenesis.

**The Delorme Operation** — In 1898 Delorme announced his ideas on freeing the heart from constricting adhesions. Sauerbruch and Rehn (1913) independently carried out the suggestions of Delorme and operated upon several cases successfully. It is only within the last decade however that pericardial resection has been accepted as an actual cure for constrictive pericarditis.

The first stage of the operation is similar to the Brauer technique. The fourth, fifth and sixth rib ends and the left edge of the sternum are usually removed thus exposing the triangle of safety. While the pleura is retracted laterally the pericardium is incised and both

## CHAPTER XXIV

### THE LUNGS

#### HERNIA OF THE LUNG

**HERNIA** of the lung is a protrusion of the organ through an abnormal opening in the thoracic cage. It usually manifests itself as a sac covered by parietal pleura and lying beneath the skin. The condition is rare and may be congenital or acquired.

*Thoracic hernia* is the commonest form due to absence of the external intercostal muscles from the costochondral junction to the sternum or of the internal intercostal muscles from the costal angle to the vertebra. The largest number occur on the anterior chest wall near the sternum as the pectoralis major does not give the same support as that supplied to the costovertebral angle by the trapezius latissimus dorsi and rhomboidal muscles.

*Cervical hernia* represents the next most common type and is probably due to a weakening of Sibson's fascia. A common site for such herniations lies between the anterior scalenus and the sterno cleido-mastoid muscles. *Diaphragmatic lung hernia* is extremely rare.

According to Montgomery and Lutz in 61 various types of hernia of the lung 21 occurred in children under fifteen years of age.

**Congenital Lung Hernia.** The condition may occur in the thoracic, cervical or diaphragmatic area. The associated defects commonly present in the ribs and sternum may be due to amniotic bands, pressure of the fetal elbow against the chest wall, lack of amniotic fluid or uterine fibroids. According to Hochsinger all pneumoceles occurring in the first few weeks of life should be considered congenital in origin.

**Acquired Lung Hernia.**—*Spontaneous hernia* may result from the coughing and straining attendant upon pertussis, bronchitis or bronchiectasis in the presence of congenital absence of the pectoral or intercostal muscles, defects in the cervical fascia or distasis of the scaleni muscles. *Pathologic herniation* may follow empyema, necrotic abscess of the ribs or abscess of the chest wall. *Traumatic hernia* usually occurs from severe injuries to the chest wall or follows operations employing the technique of Isthlaender or Schede especially when subperiosteal rib resection is omitted.

**Symptomatology.** Except for the swelling which appears upon coughing or crying, congenital types are usually asymptomatic. In acquired forms the symptoms vary somewhat depending upon

the location and type of pneumocele. The onset is usually insidious, accompanied by local pain and cough. The latter generally becomes chronic, spasmodic and non-productive.

Acute traumatic types are usually marked by the more severe symptoms caused by injury to the underlying structures. Latent traumatic herniæ are characterized by an insidious onset, pain in the region of the hernia, a pulsion mass and chronic cough which ejects the lung, making the patient twinge with pain. The herniation reduces promptly as soon as the intrathoracic pressure abates.

The hernial orifice is generally easy to palpate and presents a bony or fibrous ring through which the finger may be insinuated into the chest cavity. When the hernia protrudes, grasping the lung between the fingers produces a soft crepitant sensation.

**Treatment.** Lung herniæ do not cure themselves but strangulation rarely occurs in untreated cases. Medical or surgical therapy may be instituted depending upon the type of hernia and the existing local and intrathoracic pathology. Palliative measures consist of rest and local compression with pads or elastic devices. Various types of plastic operations have been performed: rib transection, fascial repair, periosteal transplant, splitting of the rib adjacent to the hernial orifice and using the bone flap as a covering for the hiatus and ligation of the hernial sac.

### BRONCHIECTASIS

**Historical.**—Laennec first described bronchiectasis in 1826 and during the ensuing century the clinical picture of the advanced case, with extreme cough, copious expectoration of foul sputum and clubbing of the fingers, commanded the attention of both scientist and clinician. With the application of lipiodol as an opaque contrast medium by Sicard and Forestier in 1922, renewed interest in the diagnostic and therapeutic aspects of the disease occurred.

As the roentgen ray created a new era in tuberculosis, so bronchography opened a new diagnostic approach in solving bronchiectasis. An inevitable sequel was the tremendous increase in the number of early cases discovered. With the present diagnostic and therapeutic agents at hand, it is inexcusable to permit patients to advance to the stage of advanced bilateral disease.

**Definition.** Bronchiectasis is a disease of the bronchi or their smaller tributaries in which there is dilatation of the wall. The dilatations may be congenital or be acquired through infection. The former may remain asymptomatic or become infected and develop indistinguishably from the latter. Although the disease is associated with abscess formation, even though it be microscopic, it is generally considered as a diffuse involvement of the lungs in which the pathology is confined chiefly to the bronchi.



**Incidence** —Lemon (1926) reported 63 cases of bronchiectasis in 15 500 admissions of children to the Mayo Clinic (0.4 per cent). Moll discovered 0.4 per cent of bronchiectasis in 12,225 autopsy cases at the General Infirmary of Leeds. In a study of 100 cases, Garrell found that 77 per cent occurred during the first three decades, and of these, 28 per cent developed during the first decade, also 17 per cent gave histories suggesting the disease had its onset during infancy. Graham, Singer and Ballou report a 12 per cent incidence during the first decade and 28 per cent during the second. The ages given in their series of 149 cases are those of first observation although many had antecedent symptoms. In 60 cases studied by Moll (1932) the condition was present in the first five years of life in 50 per cent.

**Etiology** —Congenital types are more properly placed under the title of congenital cystic disease of the lung and will be discussed later. It is generally believed that such patients are susceptible to repeated pulmonary infections which make them good candidates for bronchiectasis. Cystic disease in itself is asymptomatic unless mechanical changes or infection supervene.

The main processes involved in the production of the acquired form are either infection or atelectasis, or both. All other factors are secondary.

**Infection** —In the numerous studies of bronchiectasis in both children and adults, it has been found that acute illnesses such as measles, pertussis and influenzal pneumonias are frequent precursors of the condition. In others a history of aspiration of a foreign body, repeated acute respiratory infections, or pulmonary symptoms from birth or early life may be elicited.

Opie, Blake, Small and Rivers describe extensive changes which take place in influenzal pneumonia, and Kaufman those which occur in the mucosa of the bronchioles in the bronchopneumonia of measles. Erb emphasizes the marked changes in the bronchi and bronchioles, including bronchial occlusion, in pertussis pneumonia. Although some of his cases may be in contrast to our conception of bronchiectasis, the writer believes that regardless of whether the etiology is primary in the lung or bronchi, the fact that the bronchi are permanently impaired, structurally and functionally, is all important. Smith has been able to demonstrate fusospirochetal organisms in cases of bronchiectasis and has produced the disease experimentally with the organisms recovered from such cases.

Actinomycosis which rarely occurs in children is usually accompanied by bronchiectasis. Longacre and Herrmann and others have produced bronchiectasis experimentally by intrabronchial instillation of certain organisms. The causal relationship between paranasal sinus disease and bronchiectasis is not clearly established, but that the two conditions coexist has been demonstrated frequently.

Just as bronchiectasis is associated with abscess formation so chronic lung abscess is always associated with bronchiectasis. That a foreign body may cause no sequelæ when removed early is well recognized but when infection supervenes bronchiectasis results even if the patient escapes extensive suppurative pneumonitis. Ochsner was able to demonstrate by lipiodol studies that 90 per cent of university students and other adults who presented a history of repeated attacks of acute bronchitis or chronic bronchitis had bronchial dilatations.

Conditions such as prematurity, marasmus, rickets and debilitative diseases lead to impaired respiratory function. The resultant retention of secretions is conducive to recurrent acute and chronic respiratory infections and to bronchiectasis.

**Atelectasis**—This is the second factor in the production of bronchiectasis. The atelectasis may be primary or secondary to infection. When primary bronchiectasis will occur if the retained secretions are infective. Atelectasis may also develop secondary to infection through occlusion of the bronchi or bronchioles by exudate. Other causes include intrabronchial tumors or extrabronchial pressure caused by enlarged mediastinal nodes, mediastinal growths or cardiac hypertrophy.

The mechanics of atelectasis, whether it occurs in a whole lung or in a small alveolus, is the same. With obstruction of the leading bronchus or bronchi there is no ingress of air beyond the point of obstruction. The entrapped air is absorbed by the pulmonary circulation with consequent collapse of the pulmonary tissue which in time causes a greater negativity of the intrathoracic pressure. Warner and Graham have experimentally produced such changes by occlusion of the terminal bronchioles.

Children are frequently the victims of aspirated foreign bodies such as pieces of toys, coins and food particles. If the foreign body is recovered early no affection of the bronchus or supplied lung tissue occurs. If allowed to remain and the obstruction be incomplete ulceration of the mucous membrane usually develops which may be followed by bronchostenosis even though the foreign object be removed. If the obstruction is complete and the foreign body remains pulmonary and bronchial infection will result if early death does not intervene.

Other factors which have been credited with important roles in the pathogenesis of bronchiectasis are pressure of retained secretions, nutritional changes in the bronchial wall, dilatation caused by the force of inspiration, neuromuscular effects and dilatation by extrapulmonary fibrosis.

**Pathology**—This concerns essentially the bronchi and their ramifications although the pulmonary tissue itself is usually involved to some degree. It might be well to describe the changes occurring

in acute bronchitis to better understand how such relatively mild infections may progress through repeated onslaughts. In acute bronchitis the swollen reddened mucosa is covered with secretions containing desquamated epithelium leukocytes and occasionally erythrocytes. The secretions may be serous mucoid or even purulent. In so called suppurative catarrh round cell infiltration occurs in the mucosa. Although the catarrhal condition does not invade the smaller ramifications of the bronchial tree in adults these smaller branches in infants and children may become involved with much less difficulty because of incomplete development. Secondary inflammation accompanying severe infectious diseases may produce a bronchiolitis and even extend into the parenchyma and produce bronchopneumonia. Should a considerable portion of the bronchiolar tree become involved severe dyspnea or asphyxial death may result. Cases of the latter type usually show atelectasis of the pulmonary tissue supplied by the bronchiole. In patients who recover if resolution be delayed or prolonged it is easily conceivable that organization of the exudate may occur and that with increased negativity in the intrapleural pressure dilatation will result.

It has been stated that in bronchopneumonia due to measles the cylindrical epithelium may be destroyed and replaced by modified non ciliated epithelium. Kaufman describes destruction of the infiltrated bronchioles with atrophy of the elastic fibers. In chronic bronchitis more extensive changes are found. The mucosa becomes thickened infiltrated granular and at times folded upon itself with an accompanying hypertrophy of muscle and elastic tissues. Dilatations of the bronchi and bronchioles may also occur. The mucous membrane which has been densely infiltrated may be replaced by fibrous tissue or modified epithelium and the mucous glands muscle elastic tissue and cartilage may then become atrophic. Thus with repeated acute or chronic lower respiratory infection it can readily be seen that irreparable damage leading subsequently to bronchiectasis may occur.

At a later stage the mucous membrane becomes denuded and is replaced by granular tissue which bleeds easily. Ultimately marked dilatations develop lined by shaggy membranes and filled with greenish yellow pus. The modified epithelium may project into the lumen. The blood vessels are also involved and may be tortuous even with aneurysmal dilatation. The bronchial wall may exhibit hypertrophy of the fibrous and elastic layers or be atrophied or destroyed. The pulmonary parenchyma in the region of bronchiectasis shows the alveoli to be atelectatic filled with exudate or organized. The dilatations are of three main types cylindrical fusiform or saccular.

**Bacteriology**—Whereas some investigators believe that specific organisms or groups of organisms cause bronchiectasis bacterio-

logic studies of the secretions of most cases show a wide variety of bacteria staphylococcus aureus streptococcus viridans streptococcus hæmolyticus and non hæmolyticus pneumococcus mucosus encapsulatus micrococcus catarrhalis fusospirochetal organisms and the influenza bacillus

Although the predominating organisms vary considerably in different series of cases complete bacteriologic studies should be carried out aside from academic reasons with the view of employing specific therapy in conjunction with other measures

**Site of Lesion**—The lesion is predominantly that of the lower lobes. In unilateral cases the left side is involved more often in the approximate ratio of 2 to 1. Percentages of unilateral and bilateral disease vary depending upon the time the patients are seen. Singer, Graham and Ballou report 83 unilateral and 66 bilateral lesions in a series of 149 cases. Thorpe reports 67 per cent unilateral in his series of children.

**Symptomatology** In children there is no typical train of events or symptoms since these depend chiefly upon the factors initiating the disease. In the new born with atelectasis there is frequently a history of intermittent or constant cyanosis, weak cry, shallow and irregular respirations and cough. If the infant survives and is not relieved of the atelectasis it usually develops a chronic cough, occasional difficult respiration and frequent so-called chest colds. During infancy and early childhood there is no expectoration because the sputum is swallowed.

When a foreign body is aspirated there may be a short asymptomatic period followed by the sudden onset of cough, often paroxysmal in character. Several days later fever develops associated with the physical signs of pneumonia and the ensuing convalescence is slow. Subsequently a history of recurrent coughs and colds at times attended with fever and in other instances without apparent systemic effects is usually elicited.

In cases of pneumonia following pertussis or measles there frequently develops a chronic cough with intermittent attacks of acute bronchitis or pneumonia. Lastly in a small percentage of cases symptoms develop insidiously without any history of antecedent acute respiratory infection.

**Cough** This is the dominant symptom in almost all cases. It is usually chronic in nature with acute exacerbations accompanied by either slight or severe systemic reactions resembling pneumonia. The cough is frequently paroxysmal and is often initiated upon arising in the morning or assuming a position which aids drainage. As the disease progresses the cough becomes more severe.

**Expectoration**—In infants and young children there is no visible expectoration because the secretions are swallowed. Later mucopurulent sputum is raised and finally it becomes purulent and pro-

**fuse** The sputum of children is usually not foul nor does it separate in the classical fashion of an upper layer with thin frothy white or yellowish-green material, a middle one of thin clear or slightly turbid, and a lower heavy purulent layer. Far-advanced cases, however, layer out and are foul, especially if anaerobes are present. Microscopic examination may show any of the following: mucus, pus, detritus, Leyden crystals, Dittrich plugs, elastic tissue, eosinophiles and even cartilage and bone.

**Hemoptysis**—It is generally believed that hemoptysis occurs more frequently in bronchiectasis than in any other chronic pulmonary disease. Ochsner states that it develops in 50 to 70 per cent of cases, but that it is seldom severe. Thorpe found it 27 times in 53 cases, and in 2 the cough and hemoptysis preceded the physical and roentgen-ray findings by several months. Farrell reported its occurrence in 27 of 100 cases. Graham, Ballou and Singer state that alarming and fatal hemorrhages are not rare. A 'dry form' of bronchiectasis also occurs with recurrent hemoptyses but without sputum. Recognition rests upon bronchography.

**Fever**—Many of the children have no complaint of fever but upon close observation exhibit intermittent or persistent low grade elevations of temperature. Exacerbations with hyperexia are caused by acute pneumonitis either near the bronchiectatic area or in previously uninvolved parts of the lung through "spilling." The more frequent and severe the reaction in the same region, the more pronounced is the permanent effect of the disease on the pulmonary parenchyma.

**Pain**—Usually no pain occurs unless the parietal pleura becomes involved. The latter process may be fibrinous, serous or purulent. Severe pain, shock or sudden dyspnea should arouse the suspicion of either empyema or spontaneous pneumothorax.

**General Status**—Although the statement is frequently made that the nutrition is usually good, careful studies reveal that many of these children are neither well nourished nor fully developed for their ages. Lassitude is rather common even in those who appear well otherwise. It is true, however, that the degree of infection is not reflected directly in the general appearance of the child.

**Diagnosis**—The diagnosis rests upon careful history, thorough physical examination, roentgen-ray, fluoroscopy, bronchoscopy, bronchography and complete laboratory studies. The type of therapy to be instituted will depend upon the specific findings.

It is essential to obtain a history regarding familial diseases and respiratory infections, especially tuberculosis. The birth history should include the type of delivery, condition after birth, cyanosis, weak cry, rickets, and the frequency, severity and duration of respiratory infections. The history of aspiration of a foreign body is, of course, extremely significant. Bronchiectasis is suggested

when frequent respiratory infections are severe enough to force the child to bed, or when the symptoms are less severe but persistent. The relationship between the bronchopneumonias of pertussis, measles and influenza has been previously emphasized.

**Physical Findings**—These are very variable. Early in the disease, and in the so-called dry type, there are often no findings. In others a localized area of râles may appear and reappear in the same region during each acute respiratory infection. As the disease progresses, the râles become more constant. The breath sounds may be normal or diminished. In the presence of atelectasis or large cavities, the sounds may be markedly diminished or bronchial, depending upon the amount of secretion present. The percussion note may or may not be impaired. It is significant that on change of position the physical findings frequently change.

**Dyspnea**—This is not an uncommon finding, and it is often accompanied by slight cyanosis. Both may be due to fibrosis of a considerable portion of the pulmonary parenchyma, torsion of the great vessels, or effects of adhesions upon the mediastinum. The dyspnea has also been explained as due in part to edema of the terminal bronchioles with retention of secretions and limited movement of the intercostal muscles. Clubbing of the fingers occurs only in patients who have had the disease for several years and in whom the process is extensive.

**Laboratory Findings**—There are no significant laboratory findings characteristic of bronchiectasis. The sputum should be studied bacteriologically for the predominant organism with the purpose of employing vaccine therapy in cases of staphylococcus or streptococcus infection, or specific therapy in cases of spirochetal or fungus infection.

**The Blood**—Secondary anemia occurs commonly. Unless there is an acute process operating, one finds either a normal white cell count and differential or slight leukocytosis with moderate elevation of the polymorphonuclear cells. During an acute episode the leukocytosis and polynucleosis are the same as occur in any acute pneumonic process. If eosinophilia is present, ecchymococcus infection should be suspected.

**The Urine**—This is normal except in cases with continuous low-grade fever or septic types in which slight albuminuria may be present. Children with albuminuria or palpable liver and spleen without albuminuria should be investigated by the Congo red test of Bennhold for amyloid disease. It is advisable to use 1 cc. of 1 per cent solution of Congo red for every 10 pounds of body weight. Retention of less than 80 per cent is not diagnostic of amyloid disease. The writer has seen the complication occur in infants and young children with pulmonary and bone tuberculosis.

**Roentgen ray** — A normal antero-posterior roentgen ray plate of the chest does not exclude the presence of bronchiectasis. It has been amply demonstrated upon this basis that at least one-half the cases will be missed because the involvement is not sufficient to cast a shadow; moreover, left-sided lesions may be hidden by the cardiac shadow. The shadow may also be of such nature as to be confused with normal vessel markings. Marked increase of the hilar markings extending toward the base, the presence of multiple circular high lights, circumscribed or diffuse shadows or increased density, or the presence of a triangular basilar shadow warrant further investigation for bronchiectasis.

**Fluoroscopy** — Before bronchography is attempted every case should be carefully studied fluoroscopically to differentiate the lesion. This is of especial value in distinguishing an atelectatic lower lobe from mediastinal effusion or mediastinitis. It also gives an indication of the mobility of the diaphragm which is often found to be limited on the involved side.

**Bronchography** — This procedure should always be done with the aid of the fluoroscope; otherwise the diagnosis may be missed or an erroneous one made. Under the fluoroscope the lipiodol is seen as it enters the bronchi. The patient may be shifted from one position to another in order to determine whether certain shadows are due to bronchiectasis or to the imposition of one bronchus upon another whereby bronchiectasis is simulated. Flat plates of the chest are not diagnostic. Under fluoroscopy the true condition can also be visualized before the alveoli are flooded or before the oil is disseminated by coughing and masks the outline of the bronchi.

A diagnosis of bronchiectasis should never be made without the confirmatory evidence of injection of iodized oil into the bronchi. It has been amply shown that lipiodol causes no ill effects even with continued use. It should never be employed, however, in cases with high fever, debility, acute pneumonic infection or in active tuberculosis. Before injecting the oil the patient should be drained as completely as possible by bronchoscopic or postural drainage.

**Methods of Injecting Lipiodol** — There are several methods: (1) **Bronchoscopic** — This is suitable for infants and young children if proper sedation with luminal and codeine has been effected. General anesthesia should be avoided if possible. With this procedure both bronchoscopy and bronchography may be performed at one sitting. Only a competent bronchoscopist should employ the technique. (2) **Intercricothyroid method** — This is applicable to patients of all ages and is especially suited to infants and young children. The cricothyroid membrane is punctured by a short straight needle or one with a curved tip. Injection of the oil should not be begun unless the plunger of the syringe will accept and expel air freely. (3) **Transglottic route by catheter** — The pharynx and tonsillar

pillars are sprayed with 1 per cent nupercaine or similar local anesthetic excepting cocaine. The larynx is then anesthetized by indirect laryngoscopy using the same solution on a swab or curved probe. When the anesthesia is satisfactory a radiopaque catheter on a curved mandarin is inserted into the larynx. The mandarin is then removed, the catheter being directed under fluoroscopic vision into either one of the main bronchi just beyond the carina. The lipiodol is then injected. (4) Aspiration method. The fauces and pharynx are anesthetized by spraying the local anesthetic while the patient pulls his tongue forward. By using a curved cannula or a syringe without touching either the pharynx or tongue the iodized oil is then injected while the patient breathes. The passive transnasal method is ideal for cooperative children with lower lobe lesions. With the tongue drawn forward and the head fully extended 3 to 5 cc. of a 1 per cent anesthetizing solution are injected into either nostril. This is repeated three times. The oil is then injected in the same manner a few minutes later. (5) Passive transoral method (Ochsner). With this method the swallowing and gag reflexes are abolished by anesthetizing the anterior pillars. Thereafter the patient with procaine hydrochloride in his mouth gargles and aspirates the solution thereby anesthetizing the remaining anatomic structures. The gargling and aspirating procedure is then repeated with iodized oil.

In all methods except the bronchoscopic satisfactory anesthesia must be obtained before the oil is injected. The latter should always be warmed and the injection performed under fluoroscopic vision. It is important to secure the confidence and cooperation of the child before carrying out these procedures. The patient is tilted toward the side to be injected and it is essential that no coughing occur during or after injection until the roentgen ray plates have been taken. After the study has been completed the patient may expel the oil by postural drainage. Nothing should be given by mouth until the anesthesia has completely disappeared.

Before surgery is even considered all the lobes on the one side and the contralateral lower lobe should be investigated. The disease may be bilateral with practically no signs on the lesser involved side. In such cases a lobectomy may be performed without abatement of symptoms or the latter may recur. In mid field lesions the upper lobe on the left and the upper and middle lobes on the right should be investigated for possible concomitant lesions. One is not always able to diagnose bronchiectasis even at the operating table.

**Bronchoscopy** — This procedure is essential in every case and may give valuable information not obtainable by any other method. It not only aids in distinguishing non-obstructive from obstructive bronchiectasis but the involved lobes may be identified by deter-



mining the bronchi which contain pus. The method also permits obtaining uncontaminated specimens for bacteriologic study. Also prior to bronchography pus may be more efficiently aspirated than by postural drainage.

**Differential Diagnosis**—Bronchiectasis must be differentiated from chronic bronchitis, pulmonary tuberculosis, unresolved pneumonia, pulmonary abscess and empyema with fistula. In *chronic bronchitis* the history may be similar to bronchiectasis. Singer, Ballou and Graham state that even a putrid suppurative bronchitis may occur without bronchiectasis. Lipiodol studies will establish the diagnosis. Serial bronchograms should also be made from time to time for comparisons. *Pulmonary tuberculosis* is usually apical whereas bronchiectasis is a basilar disease. The demonstration of tubercle bacilli by smear culture or animal inoculation is conclusive. In infants and young children it is necessary to lavage the stomach contents if no sputum is expectorated. If the tuberculin test is repeatedly negative to doses up to 1 mg. tuberculosis may be eliminated. In cases of supposedly *unresolved pneumonia* the persistence of physical signs with or without roentgen ray findings should suggest bronchiectasis. In such cases lipiodol studies should not be done while there is fever or any evidence of an acute exudative process. Tuberculosis must also be considered and eliminated. Bronchographic studies should be done, however, by the sixth month after the onset of symptoms to determine the presence or absence of the disease. A similar study should be repeated six months later if the symptoms and physical signs persist.

**Pulmonary Abscess** There is usually an antecedent history of aspiration of a foreign body or some operative procedure such as tonsillectomy. In any event there is an acute onset with pulmonary symptoms. Although the roentgen ray findings are at first compatible with localized pneumonia within two weeks they usually show cavity formation with or without a fluid level. When such cases are treated properly complete recovery may occur; neglected cases almost invariably develop bronchiectasis.

**Empyema With Fistula**—The expectoration of pus may suggest bronchiectasis. The condition may occur as a complication of lung or bronchiectatic abscess through rupture into the pleural cavity. In cases of bronchiectasis the empyema is usually localized and drainage through the chest wall is seldom satisfactory. The actual pathology may be determined by bronchographic studies and the injection of a dye as methylene blue or gentian violet into the empyema cavity. The presence or absence of a communicating bronchus may thus be determined.

**Prognosis**—Cases of dry bronchiectasis who have only hemoptysis as a symptom do not necessarily progress. They do how-

ever have alarming hemorrhages and some fatalities occur. They are also liable to secondary infections. According to Moll 50 per cent succumb before puberty with the highest mortality during the first five years of life. Cases which show evidences of sepsis have the poorest prognosis. If the infection producing the paresis of the musculature of the bronchial wall is of short duration without weakening or destroying the bronchus recovery may take place.

### THE TREATMENT OF BRONCHIECTASIS

The success of any mode of therapy in bronchiectasis cannot be judged solely on the basis of symptoms. Irrespective of the degree of symptomatic improvement no patient can be considered cured if the bronchographic studies still show dilatations. Such patients are always subject to recurrent exacerbations.

The type of therapy to be employed in any case depends chiefly upon the age and condition of the patient, extent of disease and complications. It is difficult to reconcile the pathologic picture with cure by any means other than complete removal of the diseased tissue.

**Non surgical Treatment**—The measures comprise postural drainage, bronchoscopic drainage, intrabronchial lipiodol, climate, specific drugs, vaccine therapy and irradiation. Although none can be credited with cure in advanced cases they may nevertheless give the patient considerable symptomatic relief. Some of the methods are said to cure very early cases, probably of atony and slight dilatation of the bronchial wall without actual destruction of the mucosa.

**Postural Drainage**—This is the most effective measure in relieving symptoms. The drainage is simple and can be carried out intermittently or continuously without discomfort. The former may be secured by having the child bend over the edge of a bed from the hips with the head downward. If secretions are released cough is induced and the secretions are expectorated. The child may also discover that certain specific positions establish the best drainage. The procedure may be carried out as frequently as desired for periods of five minutes but not after meals. Singer has devised a special table which permits one to assume any angle and which can be used for constant as well as intermittent drainage. Constant drainage may also be performed by having the patient lie in bed in the Trendelenburg position at an angle of 10 to 15 degrees.

**Bronchoscopic Drainage**—This is unnecessary in cases which respond to postural drainage except when associated with tumor or other obstruction which requires bronchoscopic removal. Although bronchoscopic drainage is more effective than simple postural, the latter should be employed as adjunctive treatment. Clarf

believes that the most effective results of bronchoscopic drainage are obtained in children. It is especially efficient when paracardial triangular shadows are present with little or no bronchiectasis.

**Intrabronchial Lipiodol**—Although there is no bacteriologic evidence to prove that lipiodol is bactericidal, cures have been reported in a small percentage of early cases and relief in further advanced types. The effects of lipiodol may be due to its iodine content or the displacement of infected secretions by the oil. Ochsner in treating 161 cases noted that the number of organisms in the sputum decreased and that 3 per cent were cured by lipiodol studies and 20.5 per cent improved. Sicard and Forestier, Ochsner, Pritchard and others have reported that no serious ill effects were observed following several thousand injections of lipiodol.

**Climate**—In certain early cases climate may be of value in conjunction with other types of therapy, especially when the streptococcus is the offending agent.

**Specific Drugs**—Infants and young children rarely show fusospirochetal organisms in the sputum due to absence of gingivitis, caries and other mouth affections in which these organisms first develop. It is advisable, however, to give a course of neoarsphenamine or sulpharsphenamine if fusospirochetes are present, although a cure should not be anticipated. The drug finds its greatest efficacy in putrid abscess of the lung caused by spirochetal organisms. Neoarsphenamine is given intravenously, 10 mg. per kilo body weight every five days for six to eight doses. Sulpharsphenamine is probably less effective but can be given intramuscularly, 20 mg. per kilo every five days for six to eight doses.

In mycotic infections of the lungs, potassium iodide may be given orally using a concentrated solution, 1 grain per minim, beginning with 3 minims daily and increasing the dose to the point of tolerance. Smith suggests that if iodides are ineffective they should be supplemented by ethyl iodide inhalations, 0.5 to 1 cc. daily, gradually increasing the dose to 2 cc. three times daily.

**Vaccines**—The intracutaneous and subcutaneous use of vaccines has been generally disappointing in bronchiectasis. Experimental evidence indicates that intrabronchial injections of staphylococcus and streptococcus vaccines, antivenous and bacteriophage produce specific opsonins and agglutinins in normal animals. Little and Cannon in their studies were able to show that a higher immunity was obtained by intrabronchial injection than by subcutaneous or intracutaneous methods. Kolmer prefers using mixed vaccines, the organisms of which have been killed by 0.5 per cent cresol rather than by heat, by the intrabronchial route after bronchial lavage.

**Irradiation**—Roentgen ray has not been used extensively in the treatment of bronchiectasis. Burck has had symptomatic success

in treating fairly advanced cases in that the degree of expectoration has been reduced considerably. The type and extent of bronchial dilatation was unaffected.

Other procedures such as inhalation, thirst cure and bronchial lavage have been found to be either useless or without special advantage.

**Surgical Treatment**—What has been said for medical treatment in regard to cure applies to surgical measures; the ideal therapy being the complete removal of the diseased tissue. Whereas other forms of treatment may give symptomatic relief, the results are seldom permanent since the pathologic lung tissue is not altered considerably. With recent advances in thoracic surgery, radical procedures are being more widely employed with encouraging results. It is an error to submit an ideal unilateral case to the grimoire of palliative measures before considering more effective surgical procedures. Valuable time may be lost whereby a unilateral process may become bilateral or involve a whole lung, thus making future treatment more hazardous and less favorable. The surgical procedures which have been employed comprise pneumothorax, oleothorax, phrenectomy, pack and plumbage, thoracoplasty, cavity pneumonectomy and lobectomy.

**Pneumothorax** The results of pneumothorax therapy have been generally disappointing for several reasons: the inability to establish a satisfactory pneumothorax space, inability to maintain pneumothorax and inability to collapse bronchiectatic bronchi even with high positive intrapleural pressure. Other objections are the danger of kinking a bronchus and increasing the probability of extension of the disease, the necessity for frequent refills, and the danger of producing pyopneumothorax and bronchopleural fistula which are extremely serious complications.

Pneumothorax is used by many thoracic surgeons, however, as a preoperative measure in lobectomy or pneumonectomy for the following purposes: to accommodate the pulmonary and circulatory functions before removal of the diseased lung; to express secretions before operation and minimize the danger of spilling to the good lung during operation; and to facilitate the removal of the diseased lung. Caution should always be exercised in producing pneumothorax; the needle should be inserted over an area of lung not involved and large quantities of air should not be given at one time.

**Oleothorax** Although oily substances such as gomenal and paraffin oil have been employed in tuberculosis to maintain a pneumothorax space which may become obliterated by adhesive pleuritis or fixation of the mediastinum, their use in bronchiectasis has not been extensive. The lack of evidence that compression is greater than with air and the generally poor results of pneumothorax in

bronchiectasis do not justify their use. The dangers are paraffinoma, bronchiopleural fistula, pleuro-cutaneous fistula, oil embolism, and difficult visualization of the underlying lung.

**Phrenectomy**—Removal of the phrenic nerve may aid drainage by altering the axis of the bronchiectatic bronchi to a more horizontal direction through elevation of the diaphragm. Collapse of the bronchi by this method, however, is impossible. The danger of phrenic exceresis has been suggested by Carlson, Ballou, Wilson and Graham who have shown, by animal experimentation, that although functions are not entirely impaired, there is a delay in expectoration of lipiodol and foreign bodies from the lung, and cough is less efficient on the treated side. Fine and Starr, however, conclude that paralysis of the diaphragm has no effect on the effectiveness of cough. In any event clinical results following phrenectomy are generally disappointing. Although the cough may improve, sepsis and dilatation persist.

Some surgeons prefer phrenic exceresis as a preoperative measure to minimize the interfering action of the diaphragm during lobectomy. Others sever the phrenic nerve where it lies on the mediastinum when the chest cavity is open. *A phrenic operation should never be done in a dyspnoic patient.*

**Pack and Plumbage**—Case reports are insufficient for any definite conclusions concerning this type of therapy. The same objections to other types of collapse therapy apply here.

**Thoracoplasty**—A small number of adults and a few children have been subjected to this method of therapy. Hedblom advises removing all the ribs from the third or fourth to the eleventh close to the paravertebral edge to secure good collapse of the lower lobe without compressing the upper. He believes it to be a good procedure in early unilateral disease, especially when small peripheral bronchi are involved. Almost complete disappearance of the dilatations may occur with marked fibrosis of the lung. He does not recommend the procedure, however, when the larger bronchi are involved or in septic cases. The objections to this treatment in children are that the operative risk is as great as in other more suitable procedures, extreme deformity results, and the diseased lung remains.

**Cautery Pneumonectomy**—This procedure makes a closer approach to the ideal treatment than any of the surgical measures suggested thus far in that it removes the diseased tissue. It has been used in children with comparatively little shock and small risk. The procedure was first carried out by Graham in 1923, and Graham, Singer and Ballou state that it is most suitable in cases with abscess and bronchiectasis, and in those 'with unilateral disease who have not responded well to simple forms of therapy and for whom thoracoplasty is either not indicated or has failed to relieve the symptoms,

and upon whom the performance of lobectomy although desirable is unwise or technically impossible. The procedure can be employed even in septic cases. Graham and his co-workers do not advise its use in patients with extensive sacular dilatations or in those with bilateral disease.

The field is exposed by subperiosteal resection of two or three ribs for a distance of 6 to 8 cm. over the involved site. Unless the visceral and parietal pleurae are adherent adhesions should be produced by suture or through the application of an iodized gauze pack for ten days. The cauterization is performed with a soldering iron heated to red heat. At different sittings excystation is made into the lung tissue. If an old drainage tract exists it is used to begin the cauterization by plunging the hot iron into the sinus and cauterizing eccentrically. In cases in which the condition is chiefly that of a chronic abscess the entire abscess cavity together with smaller communicating abscesses may be destroyed or at least the roofs of the abscesses be removed. Multiple drainage openings are thus provided by exposing a large cross section of the bronchial tree. It is best to burn over a large surface area instead of burning too deeply into the lung. After the slough separates many draining fistulae may exude pus. Repeated cauterizations should not be performed oftener than every three weeks as considerable healing often follows adequate drainage. The question of how much and how often to cauterize is determined by the patient's progress. Although several cauterizations are usually required in some instances one suffices. Should hemorrhage occur from a large vessel the cauterization should be stopped and the wound packed with iodoform gauze. A non inflammable anesthetic such as nitrous oxide should be employed.

The procedure is not attended by shock. About the third day the patients develop slight fever and malaise due to intoxication from the burnt tissue and these symptoms persist until the slough separates about the tenth day.

Bronchopleural fistulae often occur after cautery pneumonectomy. Most close spontaneously or are of so little consequence that the patients refuse surgical treatment for their closure. Permanent fistulae are much less common in children than in adults.

**Lobectomy.** This is undoubtedly the ideal procedure in suitable cases. The statement that children are able to withstand lobectomy better than adults seems to be borne out by recent reports in which the postoperative course is comparatively uneventful and the mortality figures relatively low. Archibald states that the risk of lobectomy is proportionate to the sepsis present.

Our knowledge of the type of case best suited for lobectomy is still inadequate. Although most observers are agreed that bronchiectasis involving one lobe is the ideal type Alexander Ballou

Graham and Singer advise cautery pneumonectomy if adhesions are very dense as frequently occurs with peripheral cavernous dilatations. Bohrer advises cautery pneumonectomy where there are small multiple peripheral abscesses and Janes believes that only patients with unilateral bronchiectasis free from attacks of pneumonia for a reasonable length of time should have lobectomy.

Many types of operation have been suggested yet no technic is especially outstanding. The successive stages of pneumothorax, phrenic exeresis, thoracoplasty and then lobectomy has been abandoned. It is too early to state whether or not a single or two stage operation yields the better results.

*Preoperative Measures* — Preliminary treatment such as pneumothorax to accommodate the respiratory changes which occur with lobectomy to prevent spilling and to facilitate removal of the lung has been mentioned. Some operators object to pneumothorax on the ground that empyema may occur which in itself is dangerous and may preclude subsequent lobectomy.

Phrenic exeresis is advocated to immobilize the lung during its removal and also to aid in foreshortening the chest space after removal. This may be performed before the thoracotomy by the neck approach or during operation when the nerve is seen to lie on the pericardium. In dyspneic patients it is inadvisable to perform phrenic exeresis.

During the conservative therapy foci of infection such as infected paranasal sinuses should be adequately treated. It is preferable that only one sinus be operated upon when required at a time. It is not necessary however to wait until a resistant infection is completely cured before attempting lung surgery.

Postural drainage should be carried out for several days before operation and also on the day of operation. The ideal time for surgery is late morning or early afternoon when the bronchi are probably least filled with secretions. Magill suggests the use of abundant fluid and glucose for twenty-four hours before operation.

*Anesthesia* — Cyclopropane is the ideal anesthetic for lobectomy even though it precludes the use of diathermy and cautery. Some operators employ local anesthesia and others avertin supplemented by nitrous oxide and oxygen. Tudor Edwards uses intratracheal gas oxygen anesthesia.

*Operative Technic* — *Brunn's Method* — The incision is made either between the seventh and eighth ribs or between the sixth and seventh and extends from the costal cartilages to just beyond the angle of the ribs. Sharp claw retractors are used to spread the ribs apart and the intercostal muscles are severed to the pleura. When artificial pneumothorax has been produced the pleura can be opened boldly when this has not been done a small nick is made in the pleura and the finger partly covers it to allow the lung to collapse.

slowly and gradually. The interspace is then rapidly opened with scissors traction being made above and below with large blunt retractors. A Lilienthal rib spreader is finally inserted over gauze pads and the entire chest cavity is exposed. Occasionally one or two ribs require removal for more adequate exposure.

It has been noted that early in the procedure the patient frequently becomes distressed. When this occurs the rib spreader is removed and the lung is slightly reexpanded with carbon dioxide and oxygen in nitrous oxide the wound being covered with saline pads. The pulse and respiration rapidly become normal and when the operation is resumed it is seldom that the procedure has to be stopped a second time. A survey of the chest is now made. The diseased lobe may at times be noted as somewhat contracted or having changed in color or it may appear entirely normal. The diseased lobe is grasped with lung forceps and the upper lobe if the lower is to be removed is allowed to collapse against the mediastinum and is covered with a large rubber sheet and moistened tapes. Adhesions between the upper and lower lobes and between the lower lobe and the diaphragm are separated either by sharp dissection or between clamps and all bleeding points are carefully secured. The pulmonary ligament which is sometimes well developed is cut between forceps and tied. The involved lobe is freed as far as possible toward the mediastinum and is held taut by an assistant. The entire wound and pleural cavity is well walled off with rubber film and saline tapes.

A rubber tube is placed around the root of the lung and drawn sufficiently taut to produce compression of the vessels. The tube is clamped with a hemostat. Again the tube is pulled taut and another hemostat is applied and finally a third. It may be advisable to place a hemostat on the opposite side to prevent slipping. The rubber tube tourniquets control the stump and makes easy the placing of stitches and ligatures. Clamps are now placed on the lung beyond the tubing to prevent soiling when the lung is excised. These are placed far enough away so that a wedge-shaped incision may be fashioned out of the stump preferably with the actual cautery. This method of using three hemostats produces perfect hemostasis. When the first one is removed control is still obtained and gradual lessening of the pressure on the pedicle is permitted.

The main bronchus may be divided somewhat more deeply than the vessels or it may be ligatured leaving only enough projecting to prevent slipping of the ligature. The mucous membrane is cauterized. The vessels presenting are grasped separately and ligated after which a running lock stitch of chromic gut is passed closing the pedicle from the inside. This will usually control most of the bleeding. When this is completed the first hemostat is removed from the rubber tube and if no bleeding occurs the second



one is removed. Any bleeding points are carefully ligated until the stump is entirely dry. All raw surfaces are covered by suturing the pleura over the pedicle stump. It is essential that there be no hemorrhage from the stump and no leakage of air from the bronchus.

*Shenstone's Technic*—This is somewhat similar to Brunn's with certain modifications. After the diseased lobe has been completely exposed and freed, a snare of heavy cord is passed around it as near as possible to the mediastinum and the loop is drawn tight in an instrument designed for the purpose. Shenstone especially praises this simple yet highly effective method of securing the pedicle.

After removal of the diseased lobe and pleuralization of the stump is completed, a small incision is made in the ninth interspace in front of the mid-axillary line through which the end of a long tube of about 32° F caliber is drawn, the fenestrated end of the tube is placed 1 inch from the pedicle of the resected lung and maintained in place by a plain catgut suture inserted into the summit of the diaphragm. The operative wound is then closed in layers.

The distal end of the drainage tube is placed under the surface of an antiseptic solution contained in a bottle placed 2 feet below the level of the chest. The patient is encouraged to cough. This expresses air from the pleural cavity, permits of rapid expansion of the lung and prevents mediastinal flutter. At the end of twenty-four hours continuous siphon drainage is instituted.

Shenstone employed this technic in 16 cases, with 3 fatalities. Six patients were cured, 2 improved, 3 unimproved, the remaining 2 being in the hospital at the time of the report.

Archibald uses the Brunn-Shenstone technic with certain modifications. If the normal lung is not adherent he produces adhesions by "pleural poudrage" described by Bethune. This is accomplished by using any silicate powder (talc) with 0.5 per cent iodine. By thoracoscopy and a special return air powder blower, the upper lobe and mediastinum are powdered until it resembles a 'cake sprinkled with confectioners' sugar'. The air producing the pneumothorax is then extracted until 0.5 cm. or less is present by roentgen ray. Firm adhesions are formed in six weeks. In addition, the bronchus supplying the lobe to be removed is completely obstructed by a balloon inflated with air or liquid to prevent spilling of secretions. He employed this in 8 cases with success in seven. The one failure was attributed to improper insertion of the balloon, with resultant spilling to a normal lobe. A rubber tent and gauze pack are used to protect the normal lung and pleural cavity. To prevent fistula formation, the bronchus is ligated with silver wire and the stump carbolyzed.

*Author's Technic*—The details are in no wise original and the method has been evolved from advantageous procedures employed by others. An artificial pneumothorax space is effected three weeks

before the thoracoplasty and the latter is performed under avertin (basal) and cyclopropane intratracheal anesthesia.

The incision which is I-shaped begins between the vertebral border of the scapula and the spinous process at the level of the fourth dorsal vertebra and extends downward to the eighth rib and then outward following the rib anteriorly to the costal cartilage. Rack retractors separate the soft tissue and the eighth rib is removed from the tip of the transverse process to the anterior axillary line. The sixth, seventh and ninth ribs are fractured at or near the transverse processes and 1 inch of each rib is removed to prevent friaging. The pleural cavity is then opened. The interlobar fissure is separated with small sponges upon many quito forceps, scissors being used to sever any adhesions. The pulmonary ligament is ligated with suture ligatures four to six in number. At the base of the interlobar fissure the bronchus is transfixed with double No. 2 chromic catgut which is cut at the needle thereby making two sutures. These are tied one above and one below the bronchus. Another similar suture is passed through the bronchus and cut at the needle but not tied. A Nel on tourniquet with No. 2 double chromic catgut is now placed around the base of the lobe and tightened. The second suture which was passed through the bronchus is then tied distal to the tourniquet a second Nel on tourniquet of copper wire is placed its tendency to slip being prevented by applying Kocher clamps to the lung substance.

The lobe is divided near the copper wire tourniquet with a scalpel and the stump is carbolyzed and touched with alcohol. The bronchus is suture-ligated twice using an X or mattress suture. The rest of the stump is suture-ligated with four or more chromic sutures. The first chromic tourniquet is released and is now used as a suture being tied in the groove which it originally made. The sides of the stump are folded over in an attempt to pleuralize it. A No. 16 catheter is inserted through the ninth intercostal space in the posterior axillary line along the costovertebral gutter to the apex of the space and held in place by being tied with the suture that was passed through the parietal pleura in the costovertebral gutter. Another catheter is passed horizontally through the ninth intercostal space in the anterior axillary line running posteriorly over the diaphragm and held in place by a suture passed into the diaphragm. The chest wall is closed with pericostal sutures and a continuous suture of the pleura. The muscles and skin are approximated in the usual manner. Upon the patient's return to bed the catheters are inserted into a bottle which has a negative pressure of about 2 cm. of water.

The upper lobe may be removed in the same manner except the fourth rather than the eighth rib is resected.

## LUNG ABSCESS AND GANGRENE

The pathogenesis of pulmonary abscess is often a complicated phenomenon. Many factors may be operative, such as aspiration of foreign material, embolism blocking of a bronchus, and entrance into the lung of certain organisms of which various anaerobes seem to be the most important.

It is difficult to determine the incidence of the disease because (1) many patients recover spontaneously and the condition is diagnosed as pneumonia, and (2) chronic abscesses in which secondary manifestations overshadow the primary disease are often labeled pulmonary tuberculosis or bronchiectasis.

**Etiology.**—The most common initiating factors are (1) Operations upon the mouth and upper respiratory tract, (2) aspiration of foreign bodies, and (3) pneumonia.

In German's collective series of 813 cases of lung abscess, 32 per cent followed tonsillectomy. Moore estimates that approximately 1 case occurs in every 2500 to 3000 tonsillectomies. Perhaps the incidence is lower in children than adults because the anesthesia is not so deep.

Foreign bodies are a common cause. Whereas inorganic substances are easily detectable by bronchoscopy and roentgen-ray, organic substances are frequently not visualized and may become so disintegrated as not to be recognizable when bronchoscopy is performed. They usually induce rapid and fulminating infection unless promptly removed.

It is probable that most cases believed to be due to pneumonia are actually pneumonitis secondary to the aspiration of a foreign substance which is not detected bronchographically, radiographically or even at autopsy. Hartwell concludes that the pneumococcus plays no part in the genesis of lung abscess. *Streptococcus pneumoniae*, however, may produce multiple lesions.

**Pathology.**—The lower lobes are involved much more frequently than the upper, and the lesion is usually on the right side. This is accounted for by easier aspiration into the right lower lobe bronchus by virtue of its more vertical course, or by embolism due to the direct path of the pulmonary artery to the right lower lobe. The abscesses may be single or multiple, acute or chronic, and be located at any situs between the hilum and the periphery. They may vary in size to even 10 cm. in diameter. When multiple, the lesions may occur on one or both sides.

A solitary acute abscess is usually spherical and the gray or yellowish red mass is surrounded by edematous and hyperemic lung tissue. The abscess itself finally undergoes necrosis and liquefaction, and the pus may be odorless or fetid, depending upon the predominating organism. Frequently several bronchi communicate

with the cavity. As the abscess ages the edema and hyperemia subside and the wall becomes more defined by connective tissue. The surrounding pulmonary zone exhibits fibrosis and destruction of the respiratory epithelium. Bronchiectasis is usually associated. The abscess content may ultimately become thick and cheese like in consistency.

When multiple abscesses occur they are usually small but may become confluent. The changes are essentially the same as in the solitary type. In cases due to sepsis all stages of abscess formation are seen. The patients seldom survive long enough for fibrosis to develop.

Gangrene of the lung may consist of single or multiple areas usually located peripherally. Through liquefaction and excavation the lesions present a soft cavity wall with grayish yellow shaggy lining. In other respects they resemble the pathology of abscess.

Diffuse gangrene may involve a major portion of a lobe or the whole lung. The process may result from the spread of acute diffuse gangrenous pneumonitis or be due to plugging of a large bronchus by anaerobic material.

**Bacteriology**—The causative organisms are members of varied and unrelated groups. The most common comprise *Streptococcus hemolyticus*, non hemolyticus and viridans, *Staphylococcus aureus* and albus, *Pneumococcus*, *B. influenza*, *Micrococcus catarrhalis*, fusospirochetal organisms and diphtheroids. In cases of actinomycosis, amebic abscess and echinococcus cyst secondary infection usually becomes superimposed.

**Symptomatology**—With an antecedent history of aspiration or operation there is generally a lapse of several days before symptoms develop and in some instances the period is extended even to months. The chief complaints are fever, chilliness or chill, cough and localized chest pain. In gangrene or putrid abscess blood-streaked sputum or frank hemoptysis is common. Cough which at the onset is usually dry, irritating and non-productive becomes more severe as the disease progresses. There is no expectoration until the abscess ruptures into a bronchus when frank pus is ruled. If healing occurs all symptoms disappear. A decrease in the amount of sputum however is no definite index of healing. The material raised may be odorless or fetid, the latter indicating anaerobic or fusospirochetal infection. The colon bacillus may produce an equally offensive odor. When bleeding occurs the sputum is usually only blood-streaked but frank or even fatal hemorrhage may occur.

At the onset the constitutional symptoms are outstanding. Fever is high,  $102^{\circ}$  to  $104^{\circ}$  F. and septic in character. Pulse and respirations are increased but dyspnea is less marked than in ordinary pneumonia. Toxemia is moderate to severe. When the abscess ruptures into a bronchus there is a rapid fall in temperature, pulse and respi-

ration with corresponding subsidence of the toxemia. If drainage be incomplete low grade fever persists often with exacerbations.

In young children the onset simulates pneumonia. The condition is often so diagnosed until it is found that the area of the pneumonic process persists, the toxemia and fever continue and the sputum becomes progressively more purulent. In infants the sputum is usually swallowed and may only be obtainable by gastric lavage. Peripheral abscesses often suggest empyema.

Chest pain occurs only when the abscess is peripheral and involves the pleura. The location is significant since it indicates the area of lung involved. Of equal import is the fact that the same area is usually tender to pressure. In some cases pain is the dominant symptom from the onset. This occurs most frequently with anaerobic infections which are generally located at the periphery.

**Physical Signs.** These are extremely variable being influenced by the location of the lesion, communication with a bronchus, position of the patient and the amount of pus present. Centrally located cavities seldom present findings. The signs in more peripheral lesions vary from slight impairment of resonance and diminution of breath sounds to those of frank consolidation. Signs of cavitation are rarely obtained. In chronic cases clubbing of the fingers may occur.

During the acute stage there is nothing which distinguishes pulmonary abscess from acute lobar or bronchial pneumonia. The white count varies from 15,000 to 30,000 with a predominance of polymorphonuclear cells and a shift to the left in the Schilling count. During the chronic stage the leukocytes may be normal or slightly increased with mild polynucleosis. The urine often shows traces of albumin and hyaline casts. Persistent albuminuria during the chronic stage is suggestive of amyloid disease.

The sputum in all cases should be investigated for the usual pyogens by smear and culture. Other organisms to be considered are the fusispirochete, tubercle bacillus, blastomyces, actinomyces and entamoeba histolytica. The presence of elastic tissue, lung fragments or cartilage is not pathognomonic of the disease.

**Fluoroscopy and Roentgen ray.** Roentgenograms are an indispensable aid in establishing the diagnosis and in following the progress of the disease. Fluoroscopic and roentgen ray studies should always be made in the upright position. This is essential to discover fluid levels which are particularly pathognomonic of the disease. If studies are made in the supine or prone posture the contents of the cavity will not establish a definite fluid line but will produce a pattern of localized pneumonitis or a homogenous shadow which cannot be interpreted as fluid. Fluoroscopy is a valuable adjunct in that the patient can be shifted in various positions.

Early in the disease and until drainage has occurred there is seen

an area of consolidation of variable size and irregular in outline an extent. This may be confused with pneumonia, atelectasis, tuberculosis, empyema or infarct. During this stage it is often difficult to arrive at a conclusive diagnosis and herein lies the importance of serial films.

When liquefaction and drainage have occurred a cavity with fluid level is generally exhibited. When no fluid level is discernible bronchography may establish the diagnosis. It is at times difficult to differentiate peripheral lung abscess with a fluid level from localized pneumothorax. An interlobar empyema may likewise simulate abscess before drainage has occurred. Encapsulated pyopneumothorax in an interlobar fissure is also often indistinguishable from a lung abscess cavity. In such cases bronchoscopy is necessary for diagnosis.

During the course of the disease various types of shadows may be seen. In some cases there is complete healing without the occurrence of a fluid level whereas in others there is considerable surrounding inflammatory reaction with a small cavity. Changes in the size and extent of the surrounding infiltration may also occur without any effect upon the cavity or the latter may disappear and the infiltration increase. No case should be considered cured regardless of the symptomatology and clinical course until the cavity and all evidences of infiltration have disappeared. In some instances healing occurs without any trace of previous infection while in others a localized area of fibrosis persists. Chronic abscesses produce considerable fibrosis.

**Complications.** *Empyema.* In any case of lung abscess in which sudden collapse, increase in fever, toxemia or chest pain occurs empyema should be suspected. Although centrally located abscesses and those near the hilum seldom rupture into the pleural cavity it is not uncommon with lesions involving the periphery. When adhesions are present between the pleural surfaces the empyema or pneumothorax becomes encapsulated, in other cases massive pyopneumothorax occurs. Needle exploration through the chest wall is definitely dangerous.

*Extension of Disease.*—This occurs by spilling of the drainage from one bronchus into another. The spill may involve another lobe or even the opposite lung.

*Bronchiectasis.* The complication is almost always found in cases of long standing chronic abscess. Its development is more rapid in those due to microbial infection or the aspiration of organic foreign bodies.

*Hemoptysis.* Bleeding is not uncommon. The sputum may be reddish brown, blood-streaked or frank hemoptysis may occur. In exploring an abscess cavity care must be exercised not to tear trabeculations as they frequently contain vessels.

**Mediastinitis** —The condition results from suppuration and rupture of mediastinal lymph nodes. Graham and his co-workers state they have seen cases in which suppurative nodes subsided following drainage of the pulmonary abscess.

**Other Complications** —The two most common are metastatic cerebral abscess and amyloid disease. The former may occur at any time during the course of the disease. Amyloid disease is only associated with chronic lung abscess of long duration.

**Treatment** —There are no dogmatic rules for treatment. One must consider the etiology, the general condition of the patient and the clinical course.

The acute stage of the disease is arbitrarily limited to three or four weeks. If healing is not complete after twelve weeks the process is considered to be chronic. It is improbable that any abscess which has not healed by that time will resolve spontaneously. Lesions located near the hilum are most amenable to cure whereas those situated at the periphery, even though rupture occur into the bronchus, are seldom adequately drained without surgical intervention.

During the early acute febrile stage only conservative measures are indicated. These consist of bed rest, diet, drugs, postural drainage and later, bronchoscopic drainage and pneumothorax.

**Bed Rest** —Children may be kept at absolute bed rest for months if no laxity is allowed and an abundance of sympathy is exhibited. Concessions such as bathroom privileges should not be made since they are often taken advantage of and thus defeat this important element of the therapy. High fever may be combated by ice-caps and sponge baths. Fluids should be forced and in young patients it may be necessary to supplement the oral intake by cluses or infusions of saline solution with glucose. In patients who are very toxic, repeated transfusions are especially beneficial. Absolute bed rest should be maintained until complete healing is established.

**Diet** —The caloric intake should be high with more than the minimum protein requirement since there is frequently a negative nitrogen balance.

**Drugs** —The promiscuous use of cough mixtures is to be condemned. If the cough is particularly distressing the medication should contain only enough sedative to make the patient comfortable without interfering with the cough reflex and expectoration. In cases of anaerobic infection (*fusospirochetal*) the early use of arsenicals is indicated. The drug of choice is neo-arsphenamine administered intravenously in doses of 10 mg. per kilo of body weight every third day for six or eight doses. If it is undesirable or impossible to administer intravenous therapy, sulpharsphenamine may be given intramuscularly, the dose being 20 mg. per kilo. During the period of convalescence it may be advisable to administer hematines and liver extract to combat the anemia.

Vaccine therapy for staphylococcus and streptococcus infections of the lung has been rather disappointing. The experimental work of Tuttle and Cannon suggests that a higher degree of immunization is obtained by intrabronchial instillation of the vaccine than by other methods. Whittmore employs bacteriophage with good results.

**Postural Drainage** — This procedure combined with absolute bed rest often leads to spontaneous cure. Graham reported an analysis of 40 cases in children under thirteen years of age or in whom 21 (52 per cent) became symptom-free after postural drainage and



as necessary to maintain this space determined by daily fluoroscopy. The amount of air given is never massive.

**Operative Procedures** — Unless a complicating empyema or pneumonia is present, operative procedures especially into the diseased tissue are not indicated before the eighth to twelfth week except possibly in anærobic infections.

**Phrenic Crush or Phrenic Exeresis** — The results of these procedures in the treatment of acute abscesses are not remarkable and the reported cures may have occurred spontaneously. Paralysis of the diaphragm may interfere with effective cough and expectoration and delay the use of more satisfactory measures. It is unlikely that chronic abscesses are influenced.

**Pack and Plombage** — It is the experience of many surgeons that following the removal of one or two ribs with gauze packing to produce adhesions prior to pneumonostomy drainage is often improved and certain cures result without further therapy. Dolley believes that single well localized abscesses are best treated by external drainage but that other types do badly because the expulsive force of cough is lost. He tried the procedure to be described in 14 cases of chronic lung abscess that had received the usual medical care. Thirteen were improved. The technic is as follows. The abscess and surrounding pneumonitis are localized by roentgen ray and bronchoscopy. Over the most superficial site of the abscess and in a direct line with the main draining bronchus the portions of two ribs and their intercostal bundles are removed under local anesthesia. The floor of the wound which consists of parietal pleura and periosteum is then packed tightly with gauze and the wound is closed without drainage. The chest is strapped for counter pressure in case of cough. If no infection occurs the pack is removed in fourteen to eighteen days and replaced every other day with antiseptic gauze until the regenerated ribs take the form of the compression. If adequate compression is not obtained adjoining areas of ribs are removed but the pack is used only over the original site. Good results with this procedure have also been obtained by Herbert.

Overholt believes that before compression by either plombage or regional thoracoplasty is considered the lung should be fixed to the parietal pleura to prevent parenchymal displacement. He accomplishes this by stripping the periosteum from the ribs overlying the diseased area and inserting iodized gauze between the ribs and the underlying parietal pleura and periosteum.

**Pneumonostomy** — Although acute abscesses become localized and the surrounding edema and hyperemia usually subside by the fourth or fifth week, most thoracic surgeons believe that pneumonostomy should not be performed before the eighth week, provided the general condition of the patient is improving even though the cavity persists.

The high mortality in early operations is probably due to the fact that localization of the abscess is not complete enough to allow safe entry through the surrounding edematous tissue with its decreased resistance. In cases where the fever persists in spite of localization of the abscess, it may be advisable to perform plombage before attempting pneumonostomy if operative interference appears to be indicated before the eighth week. However, cases which have persistent infiltration and fever usually do badly with any type of therapy. Neuhof and Wessler believe that in putrid abscess of the lung external drainage should be performed soon after localization of the abscess.

Peripheral abscesses are most favorable for surgical drainage; they entail the minimum operative risk and are least likely to heal spontaneously. Centrally located lesions usually drain spontaneously. When their progress is unsatisfactory the surgical indications are less definite and the operative mortality is high. In such cases it is best to employ pneumothorax early before there is marked fibrosis of the cavity wall.

*Technic of Pneumonostomy*—Care must be exercised to localize the abscess accurately. The success or failure of the operation may depend upon this. Under local anesthesia, portions of one or two ribs and the intercostal bundles overlying the abscess are removed. The exposed pleura is inspected to determine the presence or absence of adhesions. If no adhesions are present, which is usually the case in deep and central cavities, or if there is doubt regarding symphysis of the pleura, gauze wrung out in iodine solution is placed in the wound and the latter is closed without drainage. Eight to ten days later the second stage is performed. If adhesions are present or have been produced, the abscess is located by using an aspirating needle with care (Figs 147, 148, 149.) Difficulty may be experienced with chronic lesions. If the cavity cannot be located after several trials, the patient should be rechecked by fluoroscopy and roentgen-ray. Once the abscess is entered the needle is left in place. Superficial cavities may be incised with a scalpel; deeper abscesses are best opened with the endotherm knife in order to seal the surrounding tissue. The cavity is gently explored with the finger to avoid the rupture of vessels. Should hemorrhage occur it can be controlled by tamponage. The cavity is then packed with gauze surrounded by rubber dam, or with cigarette drains. If tubular drainage is employed (Penrose tube), the walls should be soft to prevent pressure necrosis. The wound usually appears clean in ten days and complete healing occurs in six to twelve weeks. In treating putrid lung abscess Neuhof and Wessler prefer narrowing the cavity, exploring all loculations, and packing them with gauze. The wound is not allowed to close until all evidence of anaerobic infection has disappeared.

The persistence of cough and sputum following operation indicates the presence of other abscesses or bronchiectasis.

**Cautery Pneumonectomy and Lobectomy.**—These procedures are indicated in chronic lung abscess with bronchiectasis or small surrounding abscesses. The surgical technic is discussed in the chapter on Bronchiectasis.

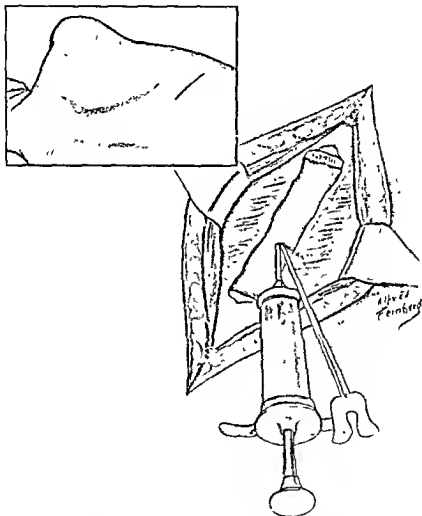


FIG. 147.—Technic of pneumonostomy for lung abscess. Grooved director is inserted into the abscess cavity after pus has been obtained by aspiration.

**Treatment of Empyema Complicating Pulmonary Abscess.**—The complication may occur spontaneously or through the injudicious use of an aspirating needle. The empyema may be localized by adhesions or be associated with air. Occasionally massive pyo-

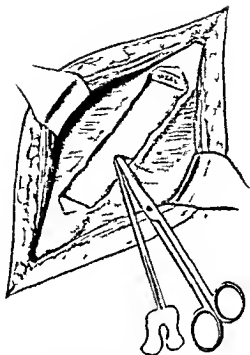


FIG. 148. Opening visceral pleura by means of special scissors with the cutting edges on the outer sides.

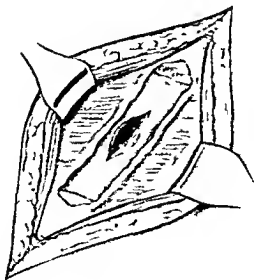


FIG. 149. Opening through lung tissue into the abscess cavity.

pneumothorax occurs. If the pus is thin it is advisable to institute closed drainage or repeated aspirations, in the presence of thick pus, open drainage is indicated. Drainage of the empyema cavity at times results in cure of the abscess but in many cases the discharge continues and a secondary operation is required to remove the diseased tissue.

## THE SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS IN CHILDREN

Pulmonary tuberculosis in children occurs in one of two forms. The vast majority are of the first infection or primary type and result from initial contact of the human organism with the tubercle bacillus. The characteristic lesion is the primary complex composed of pulmonary foci with secondary tracheobronchial lymph node involvement. In approximately 95 per cent of cases the first infection is benign and the condition goes on to definite anatomic healing with fibrosis and at times calcification. It requires no active treatment. In the remaining 5 per cent the infection progresses rapidly and caseation occurs in the lung and draining lymph nodes. Occasionally massive bronchogenic spreads develop. There is usually a rapid generalized hematogenous dissemination and in most instances death is due to tuberculous meningitis. This form of the disease will run its course despite all forms of treatment.

The second type of pulmonary tuberculosis in children is the reinfection or "adult" type. This is the result of action of the tubercle bacillus upon an allergic or previously sensitized host. Although the question as to whether reinfection tuberculosis is of exogenous or endogenous origin is a matter of considerable controversy, most authorities favor the former.

The reinfection type of pulmonary tuberculosis is comparatively rare in young children. Whereas cases have been reported at three and four years, the majority occur during the years immediately preceding or during puberty. The strain of increased physiologic processes occurring at this period is probably a predisposing factor.

In general, the treatment of secondary tuberculous infection in children is identical with that in the adult. Surgical intervention is indicated only in certain types of the disease and on that account the reader should be familiar with some of the present-day classifications of pulmonary tuberculosis.

**Classifications of Pulmonary Tuberculosis**—The classification most widely used is that of the National Sanatorium Association and National Tuberculosis Association. Briefly, the disease is divided into minimal, moderately advanced and far-advanced lesions, depending upon the extent rather than type of the process. The symptoms accompanying each stage are classified as either

none or slight moderate or severe. The classification is thus a quantitative rather than a qualitative evaluation of the disease. This is an apparent shortcoming for experience has taught that the prognosis in cases with cavity formation and positive sputum no matter how small the cavity may be is much more guarded than in patients who have extensive productive tuberculosis with or without positive sputum.

Cavitation is the source of positive sputum and patients with ulcerative lesions are a constant menace both to themselves and to others. There exists the ever present danger of the spread of infection to other parts of the affected lung or to the contralateral lung. The fact that individuals with open cavities and positive sputa may at times be symptom free should offer consolation to neither physician nor patient for the disease is characterized by periods of remissions and exacerbation and symptoms will usually reappear with progression of the disease.

The qualitative classification of Ornstein, Ulmar and Dittler offers better criteria for collapse therapy. They divide pulmonary tuberculosis into productive and exudative types, the latter being subdivided into resolving and non resolving lesions.

**The Productive Type**—This form is very slowly progressive and serial roentgen studies over a period of years reveal a slow seeding of small disseminated lesions from apex to base. Conglomerations of these lesions may occur with or without small excavations (honey combing). The symptoms and physical findings are scant in the early stages, emphysema is commonly pronounced in advanced cases. The sputum is often negative and when positive the bacilli are present in small numbers. Surgical therapy may be indicated in certain cases.

**Exudative Types**—A. **Resolving Forms** clear up entirely or exhibit scattered productive residua on roentgen ray. Surgical therapy is non indicated.

B. **The Non resolving Caseous Pneumonic Type**—It is in this form that collapse therapy is indicated. The reaction is produced in response to massive and repeated doses of tubercle bacilli in an individual with high tissue sensitivity. This is usually severe with loss of tissue, caseation, liquefaction and cavity formation. The disease may involve part of a lobe, a whole lobe or an entire lung. After sloughing occurs repair sets in and scar tissue surrounds the area with resultant contraction and distortion of the mediastinal contents. When this occurs the end stage of the caseous pneumonic process has been reached. This condition which has been designated by many investigators as fibro ulcerative or fibroid phthisis is therefore not a specific type of the disease but the end result of a previously acute stage of tuberculosis.

**Treatment**—The aim of the treatment of caseous pneumonic tuberculosis is to cause the disappearance of tubercle bacilli from the bronchial tree. In the acute phase this consists of simple bed rest and symptomatic care. This procedure allows for the differentiation between resolving and non resolving lesions.

Spontaneous pneumothorax occasionally occurs during the acute stage. When accompanied by fluid the latter should not be withdrawn unless the effects exerted by its presence are severe.

Collapse therapy is contraindicated during the acute phase of the disease. In the chronic stage however it is often of definite value.

**Collapse Therapy in Pulmonary Tuberculosis**—The prerequisites for collapse therapy in pulmonary tuberculosis are the presence of cavitation and the demonstration of tubercle bacilli in the sputum. Before entering upon its discussion it should be borne in mind that from 15 to 20 per cent of tuberculous cavities close spontaneously and any surgical measure that does not offer a higher percentage of closure cannot be considered as very successful.

**Artificial Pneumothorax**—This form of collapse therapy is employed most frequently. It should not be instituted during the acute phase of caseous pneumonic phthisis for the following reasons:

(1) It is impossible to collapse a consolidated lung. (2) there is great danger of spontaneous pneumothorax and (3) during the acute phase it is impossible to differentiate an acute benign resolving lesion from the non resolving type.

After six weeks to three months of bed rest the acute symptoms generally subside and roentgenograms may exhibit areas of absorption of the pneumonic exudate. At this time artificial pneumothorax becomes indicated.

**Diagnostic Pneumothorax**—In cases where cavitation is suspected but cannot be determined with certainty by fluoroscopic or roentgen examination the induction of pneumothorax may demonstrate definite cavity formation. Under such circumstances the pneumothorax should be maintained. If no cavitation is exhibited the procedure is abandoned. The test is of exceptional value when collapse therapy is to be instituted on one side and it is necessary to exclude cavitation in the other lung.

**Contraindications to Pneumothorax Therapy**—These comprise extensive empyema and advanced cardiorenal disease. Bilateral involvement is no contraindication and many satisfactory results have been obtained with bilateral pneumothorax. As might be expected however the best results are obtained in unilateral cases.

**Technic of Performing Pneumothorax**—Pneumothorax should be induced in an institution where the patient can be under constant observation. Any of the standard machines on the market may be employed. The patient lies upon the table with the side to be treated uppermost. A pillow is placed beneath the hips and the

six months of treatment. The procedure therefore should not be maintained too long if positive sputum persists. In instances where the sputum turns negative the pneumothorax should be maintained for at least three years before the lung is allowed to reexpand. The interval between refills is lengthened when the lesion is under control.

**Selective Collapse**—A selective pneumothorax is one in which the diseased portion of the lung is well collapsed while the healthy part remains nearly completely expanded. In such cases there is a minimal loss of vital capacity because the healthy portion of the lung is allowed to function. The procedure is of special significance in cases of bilateral collapse therapy where comparatively small changes in vital capacity may produce striking improvement in the general condition of the patient. The treatment may require small frequent refills. Serial fluoroscopic examinations are essential.

**Complications of Pneumothorax**—1 *Air Embolism and Pleural Shock* occur but rarely. Since many investigators believe the condition called pleural shock is produced by air embolism the two conditions will be considered together. They occasionally appear in patients to whom no air has been given through rupture of an alveolus by the pneumothorax needle.

The child may complain of dizziness or faintness and the breathing may become shallow and irregular and the pulse rapid and weak. He may be unable to talk and the eyes become fixed in a deviated position. In severe cases unconsciousness may occur accompanied by hemiplegia of variable duration. Should any of the foregoing appear the pneumothorax should be stopped immediately and the patient's head lowered and the foot of the bed elevated. Adrenalin or caffeine sodium benzoate may be given intramuscularly. Death rarely occurs.

2 *Serous Exudate*—Transient effusions appear at some time or other during the course of most cases of pneumothorax. Occasionally the fluid becomes purulent.

3 *Spontaneous Pneumothorax*—Spontaneous tuberculous pneumothorax is associated with the rupture of a subpleural caseous focus into the pleural space. It is usually followed by the accumulation of fluid. Spontaneous non tuberculous pneumothorax may occur from rupture of an emphysematous bleb. In such cases there is usually no outpouring of fluid into the pleural space.

With the onset of pneumothorax there occurs sudden sharp chest pain and progressive dyspnea. Cyanosis may be present also cardiac palpitation and rapid weak pulse. If unrelieved the patient may go into shock.

Physical examination reveals displacement of the cardiac apex toward the opposite side and tympany with absent breath sounds on the affected side. Upon fluoroscopic examination a marked collapse of the lung with displacement of the heart and mediastinum



to the other side is exhibited. Intrapleural pressures are usually positive.

Treatment comprises deflation of the pleural space until the intrapleural pressure becomes negative. With a large opening in the visceral pleura it may be necessary to institute continuous deflation under water.

4 *Obliterative Pneumothorax*—Occasionally the lung will reexpand and the pleural space become obliterated despite all efforts at prevention. Some authorities recommend the installation of gomenol into the pleural space in such cases.

5 *Emphysema*—This condition results from pneumothorax puncture with too large a needle. Cough and high intrapleural pressures are contributory factors. The air may collect under the skin or it may pass along the endothoracic fascia and be found over the cupola of the lung or in the neck.

*Intrapleural Pneumolysis (Jacobeus Operation)*—This procedure has proven to be of great aid in turning the sputum negative and in shortening convalescence. It is indicated in approximately 25 per cent of cases in whom adhesions are demonstrable on roentgen ray examination. Although the adhesions may stretch and the cavities close under pneumothorax therapy without interference it is best not to wait too long because of the constant danger of the spread of infection.

Adhesions are of various types. Whereas the string and cord forms are easy to sever, extensive apical cap adhesions may be most difficult to free. Since it is impossible to determine by roentgen-ray and fluoroscopy whether or not an adhesion is operable it is often necessary to perform exploratory thoracoscopy.

*Principles of Procedure*—1 There must be a sufficiently large pneumothorax space to permit instrumentation.

2 In unilateral cases only string and cord type adhesions should be severed. With extensive bilateral involvement more heroic measures must be resorted to in order to offer the patient an opportunity to recover. Too much cutting should not be done at one time however and massive adhesions are best sectioned in stages.

3 Surgery should not be attempted until three months after the onset of pneumothorax. The more the adhesions are allowed to stretch the simpler their severance will become. Moreover, by waiting, pleural effusion may be avoided. Prolonged delay, however, is dangerous.

4 Pneumothorax on the contralateral side should be induced if there is a lesion present.

*Technic of Jacobeus Operation.*—The instruments required are (1) Thoracoscope with a transformer to control illumination, (2) 2 trocars with cannulae, and (3) cautery with a transformer to control the amount of heat.

The patient is placed in the supine position with the arm of the operative side extended upward while the forearm and hand pass around and above the head. The skin is prepared with Scott's solution and the patient is draped. The electric bulb of the thoracoscope is adjusted to give a bright light and the cautery to give less than a dull red heat.

The entrance of the thoracoscope depends upon study of the roentgen ray films. The usual site is either in the second or third interspace in the mid clavicular line. A skin wheal is first made with 1 per cent novocaine and adrenalin solution and the path is infiltrated to the pleura. After the pneumothorax has been verified by aspirating a few bubbles of air the needle is withdrawn slightly and the endothoracic fascia is injected with the anesthetic solution. A 1 cm. incision is made through the wheal in the direction of the external intercostal fibers and the trocar and cannula is insinuated through the chest wall. The trocar is then removed and the thoracoscope is introduced through the cannula. The room is now darkened and careful study of the interior of the chest is made as to the number, position and type of adhesions present and whether or not blood vessels and lung tissue are present in the adhesions. The visceral pleura is also inspected for the presence of tubercles. If fluid is present and obstructs the view the thoracoscope is removed and a rubber catheter is passed through the cannula and the fluid is aspirated. The point of entrance for the cautery is chosen depending upon the thoracoscopic findings. The fourth interspace in the mid axillary line is usually selected. The method of introduction of the cautery is similar to that of the thoracoscope.

The tip of the cautery is brought into view of the thoracoscope and the adhesion is cut as near to the chest wall as possible. The cauterization is carried out slowly and cautiously with the tip constantly in view to avoid injury to the surrounding structures. The interior of the chest is finally inspected especially for bleeding. The thoracoscope is then removed and a pleural reading is taken. This should approximate zero. The cannulae are then removed and the incisions sutured with black silk. Sterile dressings are placed over the wounds and the chest is tightly strapped with adhesive plaster.

**Complications** —The following may occur: (1) Spontaneous pneumothorax. This may be due either to cauterization of the lung or the tearing of a partially severed adhesion following postoperative refill. (2) pleural reactions. (3) empyema. (4) hemorrhage. (5) obliteration of the pleural space. and (6) subcutaneous emphysema.

**Phrenectomy** —The operation is indicated in cases of pneumothorax where the cavity is suspended by an adhesion to the apical portion of the chest wall and the base of the lung is fixed to the lower mediastinal surface of the chest. The cavity is thus sus-

pended from two fixed points. The rationale of phrenectomy in such cases is that the lowest of the fixed points will be released somewhat by elevation of the diaphragm and increased amounts of air will be tolerated so that the lung can be further collapsed and the cavity obliterated.

Phrenectomy has also been employed as a single procedure in lower lobe cavities and in cases where attempts to reexpand a lung which has been under pneumothorax therapy for several years have proven unsuccessful. The rationale in the latter type is that elevation of the diaphragm will help to obliterate the pleural space. In cases where several stages of thoracoplasty fail to control lesions in the lower lung field phrenectomy may be elected.

One great disadvantage of phrenectomy is that a large amount of healthy lung tissue in the lower lobes is functionally sacrificed with resulting decrease in vital capacity. This is of special significance in advanced bilateral cases and in those in whom it is necessary to perform a thoracoplasty at some future date.

Statistics concerning the results of phrenectomy vary considerably. As nearly as can be determined closure of cavities is obtained in about 20 per cent of the cases. Since 15 to 20 per cent of all cavities close spontaneously the procedure offers nothing spectacular.

**Technic of Phrenectomy.** Morphine sulphate grain  $\frac{1}{2}$  and atropine  $\frac{1}{100}$  are given one hour before operation. A sand bag is placed between the shoulder blades and the head is turned sharply to the right or left depending upon the side to be operated. The operative field is prepared with Scott's solution. Following infiltration of the skin and deep tissues with 1 per cent novocaine and adrenalin an incision is made 2 to 3 cm. above and parallel to the clavicle beginning at the posterior border of the sterno-cleido-mastoid muscle and extending ventrad the skin subcutaneous tissue and platysma being divided by sharp dissection. The investing layer of deep cervical fascia is then split longitudinally. If the omohyoid muscle is encountered it is retracted either up or down and the transverse cervical vein or the internal jugular is withdrawn to one side. The scalenus anticus muscle is now identified with the phrenic nerve crossing it beneath the deep cervical fascia (Figs 150 and 151). The latter is divided and the nerve is lifted by a hook infiltrated with 1 per cent novocaine clamped and cut high. If exeresis is to be performed the nerve with all its branches is dissected from the fascia and twisted slowly out of its bed by a gradual rotation of the clamp (Fig 152). In 24 per cent of the cases an accessory branch is derived from the subclavius nerve.

The procedure for temporary phrenectomy is the same as the above except that all the branches of the nerve in the neck are cut and the phrenic itself is crushed by a hemostat.

The platysma is sutured with two or three interrupted plain cat gut sutures and the skin approximated by a subcuticular stitch of fine chromic or dermal suture.

**Complications**—These occur infrequently and comprise mediastinal hemorrhage, mediastinal emphysema, bronchial obstruction.

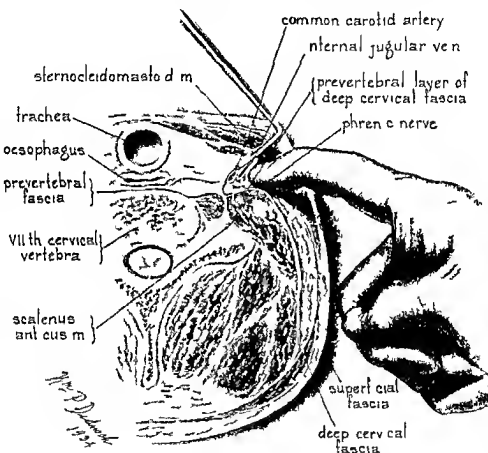


FIG. 150 Relationship of the phrenic nerve at the level of the seventh cervical vertebra.

Horner's syndrome, brachial plexus injury and death due to erroneous section of the vagus nerve.

**Paravertebral Thoracoplasty** The aim of the procedure is to obtain an almost complete obliteration of that portion of the chest cavity which overlies the diseased lung. This generally requires total resection of the first and second ribs and almost complete

resection of the remaining upper ribs. The operation should be done in several stages depending upon the extent of the lung involvement.

**Indications**—1 In the opinion of many surgeons thorico-plasty is the operation of choice in unilateral cases with cavitation above the first rib. The rationale is that a number of successful results

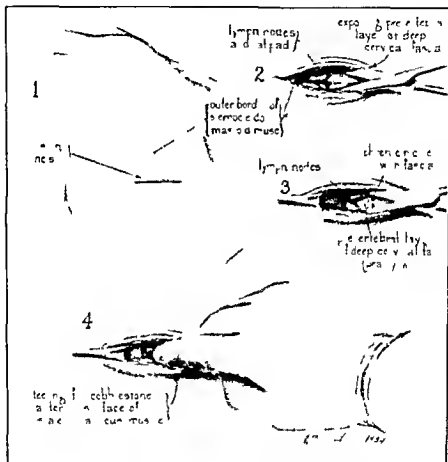


FIG. 151 Exposure of the phrenic nerve

may be obtained with a single stage operation and with relatively little deformity of the thoracic cage. The patient is also saved years of pneumothorax therapy with the possible complications of such treatment.

2 In chronic caseous pneumonic tuberculosis where other methods of collapse therapy have failed to control the disease.

3 To obliterate the pleural space in certain forms of empyema.

It has also been employed in cases where a collapsed lung will not expand

**Technic of Thoracoplasty**—Under general anesthesia preferably cyclopropane, the patient is placed in the lateral position with the diseased side uppermost. The chest rests against a rubber cushion both anteriorly and posteriorly so as to avoid undue pressure. The lower leg is bent at right angles at the knee and the legs and feet are attached to the table by a belt. The incision, beginning at the level of the second dorsal spine about 3 cm. above the spine of the scapula, is curved downward and forward to terminate in the

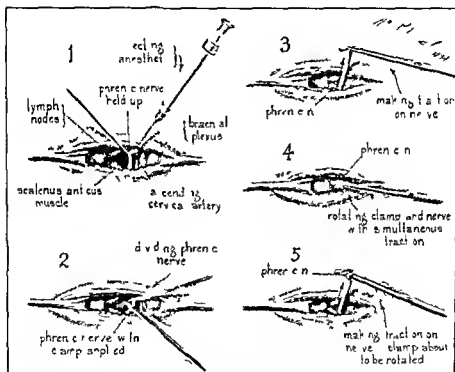


FIG. 152.—Phrenicectomy

posterior axillary line 3 cm. below the inferior angle of the scapula. The skin and subcutaneous tissue are sectioned and the fascia is incised. Beginning at the triangle of auscultation, the trapezius and rhomboids are divided, followed by the latissimus dorsi. Meticulous hemostasis is maintained throughout. The scapula is then retracted and the connective tissue between the anterior serratus muscle and the chest and scapula is pushed aside. The muscle is then cut at its costal origin. Subperiosteal resection of the third rib is performed first. The second rib is next resected and then the first. The periosteum of the latter is incised on the outer

border above and outside the muscles of the first intercostal space in order to preserve the intercostal vessels. The inferior surface of the rib is first cleared of its periosteum and the superior surface is then stripped working from the posterior toward the anterior end with a sharp elevator which is not allowed to lose contact with the bone. By placing the left index finger in front of the advancing elevator the danger of injuring the subclavian vessels is avoided. The first rib is now ready to be resected. As soon as the rib is sectioned it is grasped with bone forceps and a gauze pad is placed between the cut end of the rib and the brachial plexus. The periosteum remaining on its superior and inferior surfaces is stripped and the rib is sectioned at the cartilage the posterior stumps being removed with a large rongeur. The intercostal muscles, nerves and blood vessels are then resected. The apex of the lung is now freed from its connective tissue attachments along the brachial plexus and the costovertebral gutter by a small sponge held in mosquito forceps (Semb technique). Any connective tissue bands encountered during the mobilization which are too strong to be stripped by sponging are clamped, cut and ligated. The apex of the lung is then gradually depressed to the level of the fourth rib and the arm and shoulder are pushed backward so that the wound edges approximate. The muscles are sutured in one layer with a continuous chromic No. 2 locked suture, the fascia with silk and the skin edges with Michel clips. A strip of gauze saturated with alcohol is placed over the incision and the wound is dressed with sterile gauze maintained in place with adhesive strip.

*Second Stage*—The operation is carried out in the same manner as the first stage. The incision is made through the previous scar and the fourth, fifth and sixth ribs are resected, also the intercostal bundles. The regenerated third rib is then removed and the apex of the lung is permitted to drop.

*Third Stage*—The incision begins in the mid axillary line of the eighth interspace and is carried through the previous scar. The seventh, eighth and ninth ribs are then resected.

**Complications of Thoracoplasty**—1 After prolonged pneumothorax therapy the loss of resiliency and the increase in thickness of the pleura may interfere with proper collapse of the lung. Also fixation at the apex often prevents the proper drop of the apical portion. This complication prolongs and increases the danger of the thoracoplasty.

2 *Postoperative Atelectasis*—The condition is due to plugging of the bronchus by a purulent secretion. The condition usually occurs within twenty-four hours after operation with marked dyspnea, rapid shallow breathing, fever, rapid pulse and fall in blood pressure. Preventive measures comprise preoperative drainage of the bronchi by cough and postural drainage and the aspiration of secretions.

through an intratracheal catheter during the operation. When symptoms appear treatment consists in the rapid introduction of an intratracheal catheter and thorough aspiration of the bronchial tree.

3 *Paradoxical Respiration* —In this condition the chest expands with expiration and retracts during inspiration. The phenomenon is partly due to removal of the bony support of the chest and also to bronchial obstruction and atelectasis on the affected side.

4 *Postoperative Pneumonia and Tuberculous Spread* —The spread of disease following operation is due to the aspiration of secretions from the operated to the other side.

5 *Air Embolism* —This complication is rare.

6 *Postoperative Shock* —The condition is treated by infusions of normal saline and glucose or by blood transfusion.

7 *Scoliosis of the Spine* —This complication is very important in children. In some instances the deformity is so marked as to disable the child to a considerable degree. The condition may be prevented by early orthopedic care through the application of a brace or cast to the chest as soon as possible following operation.

8 *Fixation of the Scapula* —The condition may be prevented by early mobilization of the arm on the operated side. If fixation is due to osteomyelitis of the scapula or rib the involved area should be resected.

9 *Persistent Pain Following Thoracoplasty* —This is usually caused by intercostal nerves becoming ensnared in fibrotic tissue or regenerated bone. Treatment comprises an attempt at nerve block by injection of alcohol or paravertebral resection of the regenerated bony plate.

10 *Postoperative Bronchiectasis* —The treatment for this condition is palliative. (Refer to Bronchiectasis.)

11 *Revision of Thoracoplasty* —This is indicated in instances where the thoracoplasty has failed to collapse cavities. The procedure is to resect the regenerated ribs.

**The Treatment of Tuberculous Empyema** —The treatment of empyema complicating tuberculosis is still controversial. Various dyes and other substances such as gonadal have been injected into the pleural cavity without remarkable results.

The simple type with negative sputum is best left alone until the amount of fluid becomes great enough to embarrass respiration. Under such circumstances enough fluid should be withdrawn to relieve symptoms.

Tuberculous empyema with positive sputum should be drained followed by thoracoplasty at a later period.

In mixed infection types of empyema various organisms may occur in conjunction with the tubercle bacillus: streptococcus, staphylococcus, pneumococcus, *B. pyocyaneus* and anaerobes. The



sources of the infection are manifold: contamination from the outside during aspiration, fistulization of the needle tract; spontaneous pneumothorax with bronchopleural fistula, empyema necessitatis; and rupture of a tuberculous cavity into the pleural space following a Jacobus operation.

Treatment comprises immediate and adequate drainage. In empyema without bronchopleural fistula, the pleural cavity is irrigated every four hours with 1 to 5000 azochloramide until the washings are clear. In the presence of a bronchopleural fistula, warm saline solution is injected every four hours until the washings are clear. In the interval between irrigations the end of the drainage tube is left open.

In cases of anaerobic empyema the patients are often desperately ill and dyspnea and cyanosis may be marked. A large needle inserted above the fluid level will relieve the intrathoracic tension. Following this, closed drainage is instituted and the pleural space is irrigated with warm saline solution every four hours. The patients are benefited by daily infusions of glucose in saline and repeated blood transfusions.

The above procedures are all preparatory to thoracoplasty. The latter should be performed as early as possible and in two or more stages. If the pleura is extremely thick, it will be necessary to perform a Schede operation.

**Schede Operation**—A multiple stage thoracoplasty is performed in the same manner as described under Operative Technique for Thoracoplasty except that the thickened parietal pleura covering the cavity is resected. Careful hemostasis must be maintained during the deroofing of the parietal pleura as extensive bleeding often occurs. A few layers of washed iodoform gauze are then placed in the cavity and the soft tissues of the chest wall are allowed to fall therein. The wound is closed by interrupted dermal sutures.

**Bilateral Caseous Pneumonic Cases**—As previously mentioned, the presence of bilateral disease offers no contraindication to collapse therapy. The value of diagnostic pneumothorax has already been stressed.

**Bilateral Apical Cavities**—In these cases bilateral pneumothorax should be tried. If the sputum remains positive with a satisfactory collapse, thoracoplasty should be done on the more advanced side. If neither side is well collapsed, bilateral thoracoplasty may be attempted.

**Unilateral Apical Cavity and Extensive Disease on Opposite Side.**—A thoracoplasty should be done on the more advanced side and pneumothorax induced on the contra side. If a pneumothorax can be performed on the worse side but not on the other, thoracoplasty should be attempted on the less advanced side.

*Extensive Bilateral Lesions*—Most cases are unfit for surgery. If the patient's general condition is suitable surgical measures may be attempted.

### HYDATID CYSTS OF THE LUNG

Echinococcus disease is rare in North America especially among the native born. It is relatively common in Iceland, Australia and the Argentine where there is close contact with infected dogs or where the latter are used for herding animals, the animals becoming the intermediate hosts through eating contaminated herbage.

The liver is involved in 70 to 80 per cent of infected humans and the lungs in 5 to 20 per cent. Infants are apparently immune. In Morquio's series of 212 cases in children the youngest was four years. Graham and his co-workers state that lung cysts are more frequent in children than in adults and that brain cysts are seven times more common in early life.

*Pathology*—The etiologic agent is the *Taenia echinococcus* whose adult life cycle occurs in the upper intestinal tract of dogs and canine type animals. The ova excreted in the feces gain entrance into the human body through contaminated water and vegetables or by close contact with infected animals. The ovum liberated through digestion of its capsule by the gastric juice passes through the intestinal wall into the blood or lymph stream. It usually enters the portal circulation and the liver is accordingly the most common site of primary involvement (70 to 80 per cent). The lungs are usually invaded secondarily although in some instances the pulmonary lesion is the primary and sole pathology. (fig 173) Morquio believed that in his series of children the disease was primary in the lung. Dew states however that whenever an intrathoracic cyst is found to contain daughter cysts an hepatic origin should be suspected and this should lead to an investigation of the diaphragmatic region.

The lung may become secondarily involved from the liver through the diaphragm and pleura. Although the mechanism is not clear it is significant that the right lower lobe is affected most often. Metastases may also occur from the abdominal viscera to any portion of the lung.

When the ovum lodges in the pulmonary parenchyma its hooklets are lost and it forms an hydatid cyst. The wall of the latter is composed of an outer laminated elastic cuticle and an inner granular parenchymatous layer. The cyst may be uni or multi loculated and when developed to about 3 cm in diameter the parenchymatous layer gives rise to brood capsules, scolices and at times daughter cysts. Phillips believes that hydatids enlarge more rapidly in the lungs than in other organs because there is less mechanical resistance. With increasing size the cyst becomes surrounded by

fibrous tissue adventitia which may become extremely thick in old cases. The cysts contain water-clear fluid and hooklets. (The watery fluid has a salty taste.) Degeneration cysts may contain a thick gelatinous material with calcareous deposits.

The cysts may be located in the central or peripheral portions of the lung and enlarge to 10 or 12 cm. in diameter. Rupture may occur into a bronchus, the pleura or pericardium. Centrally located cysts are most likely to rupture into a bronchus and spontaneous



FIG. 153. Echinococcus cyst of the lung in a boy of nine years. Localization of 1 per cent. complement fixation test 4+. Cyst enucleated with ease.

hemothorax may follow. In cases of incomplete drainage secondary infection generally develops.

**Symptomatology.** Intact cysts produce no constitutional reaction and the symptoms are chiefly those of pressure upon the bronchi and vessels. Centrally located cysts may remain asymptomatic for long periods.

The most common symptom is dry hacking cough. This may be extreme and almost incessant when the cyst rests upon the

diaphragm. Hemoptysis occurs in the majority of cases. In the early stages it is usually slight but when rupture occurs the hemorrhage may be alarmingly copious. Some patients have a sense of intrathoracic weight and oppression. Dyspnea is variable. Pain does not usually develop unless the pleura is involved.

Spontaneous rupture into a bronchus occurs frequently. The catastrophe is attended by severe paroxysmal cough, dyspnea, pain and profuse expectoration of salty tasting watery fluid. The patient may feel that the chest is being flooded. Some of the fluid may escape into the blood stream and produce an anaphylactic reaction manifested by localized or generalized urticaria, nausea, vomiting, diarrhea, abdominal pain, dyspnea, cyanosis and even profound shock.

The expectorated material frequently contains hooklets and portions of the cyst wall which resemble grape skins. Evacuation of the cyst may occur immediately or take several days or it may empty incompletely and drain intermittently for weeks. In the latter instance secondary infection generally occurs producing signs of sepsis with purulent sputum.

When rupture occurs into the pleural cavity there is usually sudden pain with marked dyspnea and collapse. Rupture into the pericardial space causes severe pain and circulatory distress.

**Physical Signs**—Small centrally located cysts exhibit no physical signs. Large peripheral types may cause bulging of the chest and limited respiratory excursion. Diminished fremitus, dullness to flatness and diminution or absence of voice and breath sounds depend upon the amount of overlying lung tissue. Such findings at the base may be confused with those of effusion. After evacuation of the cyst has occurred the percussion note may become tympanic and the breath sounds may or may not be altered.

**Roentgenologic Examination**—Cysts filled with fluid exhibit a well-defined homogenous shadow resembling that of an abscess. Bronchography may reveal bronchial obstruction and outline the cyst wall. When rupture has occurred a fluid level is usually demonstrable. In cases where the contents are thick the level is usually rough and uneven and does not shift as readily as in abscess (Tillier).

**Laboratory Findings**—Before rupture it may be impossible to establish a definite diagnosis without a complement fixation (Ghedini Weinberg) or intradermal (Casoni) test. The latter is simpler to perform and is probably the more sensitive. The substance used is hydrated fluid obtained from cysts in animals which has been passed through a fine Berkefeld filter and kept sterile. The fluid is more sensitive when freshly prepared. Intradermal injections of 0.25 to 0.3 cc. are employed. A positive test produces an urticarial wheal with pseudopodia within twenty minutes. Occasionally

the sensitivity of the patient is sufficient to cause an anaphylactic reaction

The finding of hooklets or portions of the cyst wall in the expectorated material is pathognomonic of the disease. It cannot be stressed too strongly that diagnostic aspiration is to be condemned because of the dangers of anaphylactic reaction, rupture into the pleural space, contamination of normal lung tissue and secondary infection of the cyst.

Eosinophilia may be present but its absence does not exclude echinococcus infection. The presence of bile in the cyst fluid denotes hepatic involvement.

**Diagnosis**—Before rupture occurs the diagnosis is made upon the history of paroxysmal non-productive cough, frequent hemoptysis, absence of constitutional symptoms, radiologic evidence of a well-defined homogenous mass in the chest and a positive complement fixation or intradermal test. Following rupture the diagnosis is established by the history of sudden dyspnea and copious expectoration of salty thin fluid. In cases with secondary infection the roentgenogram of a cavity with an uneven fluid line is significant. The finding of hooklets or portions of the cyst wall in the sputum is pathognomonic.

The disease may be differentiated from basilar effusion by the globular outline of the cyst on roentgen ray. Tuberculosis is usually associated with fever and other constitutional symptoms, the Mantoux test is positive and tubercle bacilli may be demonstrable. Moreover tuberculosis rarely produces a smooth globular well defined shadow and bulging of the chest on the involved side is never present.

**Treatment**—Conservative therapy is indicated in children with centrally located cysts or with vomica as the majority recover spontaneously. Morquio states that 90 per cent of the cases that rupture are cured. Phillips agrees that central cysts should be treated expectantly but believes that peripheral types require prompt surgery.

Several methods have been employed for cystectomy. Syme and Finocchetti enucleate the cyst in a one-stage operation and close the chest without drainage. Bird uses this method when there is no thickened adventitia. After removal of the cyst Fairley obliterates the cavity by suturing the walls together. Pratt recommends a two stage operation, adhesions are produced first between the chest wall and pleura by using iodine gauze packing against the pleura, ten days later the cyst is removed and the wound closed. Of 29 cases treated by this method 27 recovered.

If secondary infection occurs the treatment is the same as for suppurative lung infection. When centrally located pneumothorax may be employed in peripheral lesions pneumonostomy is advisable. Drugs and radiotherapy are ineffective.

## NON PARASITIC CYSTIC DISEASE OF THE LUNG

There is no uniform terminology for non parasitic pulmonary cystic disease. Various names have been applied: fetal bronchiectasis, congenital bronchiectasis, atelectatic bronchiectasis, honey comb lung, congenital cystic disease of the lung, congenital cyst formation of the lung, and congenital cystic malformation of the lung. The disease was first described by Meyer (1898). Koontz reported the first case in American literature in 1925 and collected 108 histories from the European literature. Since then many cases have been observed. Wood (1934) collected 23 additional cases from the English and American literature, 7 of which were confirmed at autopsy.

**Incidence**—The disease has been found to exist in the fetus as well as at all ages. Many cases are probably unrecognized because the condition remains asymptomatic unless mechanical changes or infection occurs. In some instances the finding is an incidental one during clinical study or at necropsy. There is no relationship to sex. In Koontz series the right side was involved in 25 per cent, the left in 50, and both lungs in 25 per cent.

**Pathology** The cystic dilatations may arise from any portion of the lower respiratory tract and consist of dilated bronchi with stratified ciliated epithelium surrounded by muscle and cartilage or resemble large emphysematous bullae with thin walls lined with low epithelium. The cysts may be single or multiple, localized or diffuse and may or may not communicate with a bronchus. They may contain fluid or air or both, also mucus, desquamated cells and granulation tissue. When infection occurs the contents become purulent. Small cysts are usually lined by cuboidal or flat epithelium and the larger ones by stratified ciliated columnar. A membrana propria is always present and when the epithelium is absent the membrane forms the cavity lining. Mucous retention cysts may also be present.

**Pathogenesis**—Many theories have been advanced to account for the development of the disease. Although the cases show great similarity, it is probable that the causes are manifold. The following have been suggested: faulty development of the lymphatics in the portion of lung involved; fetal adenoma due to growth of cell rests surrounding the bronchi; faulty development of the pulmonary buds due to syphilis; persistent atelectasis after birth; bronchopneumonia causing dilatation of the terminal bronchioles and in fundibula; congenital cardiac defects by preventing expansion of the lung due to faulty pulmonary circulation; agenesis of the pulmonary buds; and interstitial or bronchogenic origin by occlusion of a bronchus, bronchiole or smaller air passage with secondary formation of retention cysts.

**Symptomatology** —The disease even though extensive usually remains asymptomatic unless infection sudden enlargement or rupture of a cyst occurs. The symptoms consisting chiefly of intermittent attacks of dyspnea and cyanosis are generally due to imperfect communication between the cyst and a bronchus. Miller, Parmelee and Apfelbach believe that a check valve mechanism is responsible for the increase in size of cysts by virtue of the fact that there is free ingress but no egress of air. Recurrent attacks of dyspnea and cyanosis should suggest the pathology especially when the syndrome is repeated for many years.

Large cysts may cause dyspnea unproductive cough cyanosis and hemorrhage. The last may occur secondary to rupture or from bleeding granulation tissue. Dysphagia may also be present. If secondary infection occurs the symptoms resemble those of any acute pulmonary infection. Purulent sputum may result.

**Physical Signs** There are no characteristic physical findings. Multiple cysts involving a whole lobe or an entire lung or large solitary cysts filled with air may produce hyperresonance and diminished or absent breath sounds and when filled with fluid dullness or flatness with absent breath sounds. Fluid cysts in or near the mediastinum may exhibit widened manubrial dullness (d'Espine's sign). Extremely large cysts may cause displacement of the heart and mediastinum.

**Röntgen ray Findings** —Single large cysts filled with air present annular shadows of diminished density outlined by a fine smooth line without evidence of inflammation. In extremely large types this line may be absent or there may be surrounding areas of increased density due to atelectasis of the neighboring lung. Cavities containing fluid may exhibit definite fluid levels. These are frequently multiple and may suggest lung abscess.

Bronchography injection of lipiodol by thoracentesis and diagnostic pneumothorax may further aid in the diagnosis.

**Treatment** —No treatment should be instituted unless there are definite and incapacitating symptoms. Spontaneous improvement occasionally occurs through communication with a bronchus or closure thereof. In large cysts with a check valve mechanism it may be necessary to institute continuous deflation. At times the removal of a few hundred cubic centimeters of air will produce relief. Although this procedure is not always effective nor without danger patients who are distressed deserve its trial. In cases where continuous deflation is indicated an attempt may be made to seal the opening by injecting oil of gomenol iodized oil or saturated glucose into the cavity. These agents have been employed with moderate success. Thoracotomy and application of silver nitrate to the bronchial openings may ultimately be required.

Infected solitary cysts should not be aspirated as adhesions are

not always present and empyema may result. It is advisable to first create adhesions by placing iodine gauze over the parietal pleura following subperiosteal rib resection. After ten days the cyst may be incised and drained with safety. An attempt may also be made to close the fistulous opening with silver nitrate.

Large multilocular cysts which become infected should be opened widely and drained. When the infection has cleared the cysts may be separated from the lung by dissection until the pedicle connecting them with a bronchus is reached. The bronchus is then severed and closed with interrupted sutures of fine black silk. The stump may be covered by neighboring lung held in place by fine catgut. Eloesser prefers to perform lobectomy after the acute infection has subsided following incision and drainage. He removed the lower lobe in 3 cases with good results. Complete pneumonectomy may be performed in unilateral cases. Cystic disease in the upper lobes usually drains well spontaneously.

### ACTINOMYCOSIS OF THE LUNG

Actinomycosis is a rare disease especially in children. Of Sanford's series of 678 cases reported in the American literature only 22 occurred in patients under ten years of age. Thoracic lesions are present in approximately 15 per cent of all actinomycotic infections.

**Etiology**—The causative organism, the *Actinomyces bovis*, is composed of non-septate branching filaments whose ends may or may not exhibit clubbing. The fungus stains irregularly Gram positive, is non-acid fast and grows only with difficulty under anaerobic conditions. In the tissues and in the purulent discharge clumps of the organisms appear as sulphur granules which vary in size from a fraction of a millimeter to several millimeters.

**Source of Infection**—It is now generally believed that contact with infected animals does not cause the disease and that infected grass or straw is not the vector. Lord states that from the clinical aspects of the disease the biology of the *actinomyces bovis* and experimental observation *actinomycosis is to be regarded as arising* in consequence of organisms harbored in the mouth by normal persons. It is probable that the organisms gain entrance into the body through traumatized tissue. Ellis believes that pneumonia and trauma to the chest wall may be responsible for the primary type of pulmonary actinomycosis.

**Mode of Entry**—Two forms are recognizable. (1) The primary caused by direct aspiration of the fungus into the lungs or by extension through the esophagus into the mediastinum, lungs or pleura and (2) the secondary type in which the thoracic structures are



involved directly from adjacent infected areas or hematogenously from distant *nidi*

**Pathology** The chronic granulomatous disease is characterized by simultaneous fibrosis vascularization and destruction of all types of tissue except the lymphatic the latter may become involved when generalized dissemination occurs. Microscopically there is marked interstitial connective tissue and granulation formation about the abscess area. Giant cell formation is absent but epithelioid mononuclear and polynuclear cells are numerous. The central area containing the fungi may be in any stage of abscess formation.

The thoracic lesions occur as firm hard bluish red areas which may or may not exhibit sinus formation into the thoracic cage. The pleura is often extremely thickened fibrotic and adherent to the chest wall and lung. It may contain numerous small abscesses and is frequently canalized through connections with the lung tissue and bronchi. The involved lung becomes firm leathery and heavy with multiple hard raised nodules whose centers usually contain pus. The bronchi may or may not exhibit peribronchial involvement and bronchiectasis.

**Clinical Manifestations**—Good uses the following classification (1) Bronchoactinomycosis in which the disease is confined to the bronchi (2) pneumoactinomycosis the lung tissue being chiefly involved (3) pleuropneumoactinomycosis both tissues being invaded by extension from one to the other and (4) thoracopulmonary the thoracic cage pleura and lung being involved.

**Site of Lesions**—Although the lesions may occur in any portion of the lung the initial sites are usually basilar and frequently bilateral. Good believes that involvement of the right lung is usually due to aspiration whereas that of the left connotes ingestion of the fungus. When aspiration occurs the lesions are first confined to the bronchi producing purulent bronchitis. Bronchiectasis with pulmonary invasion soon follows and the process gradually extends to the pleura and chest wall.

**Symptomatology**—The onset of the disease may be acute or chronic. The former type resembles an acute pneumonic infection except that resolution is extremely slow and is never complete. The cough persists. During this period the infection may resemble unresolved pneumonia tuberculosis lung abscess empyema or bronchiectasis. Cases with an insidious onset have few or no symptoms until nodules abscesses or sinuses develop in the chest wall. Pulmonary symptoms then appear and become progressive.

The most common symptoms are fever cough chest pain expectoration and loss of weight. The fever is low grade unless the onset is acute or secondary infection occurs. The cough is severe in acute cases but is usually mild in the insidious type. At first it

may be non productive then mucoid sputum appears and later frank pus. Chest pain is a common symptom due to extension of the disease to the pleura. Sinus formation and external abscesses occur in approximately 50 per cent of the cases. Hemoptysis is not uncommon even though the vessels are spared. The bleeding is never profuse and generally appears as streaking from the oozing of granulation tissue.

**Physical Signs**—These are of no aid in making a specific diagnosis. The involved lung generally shows impaired movements of the chest wall with dullness to flatness. The breath sounds are usually markedly diminished or of distant tubular character suggesting fluid or consolidation. The mediastinum is seldom displaced.

**Roentgen ray Findings**—There is nothing characteristic in the roentgenologic or fluoroscopic examinations. The shadow usually seen in a dense homogenous one over the involved area, the pulmonary lesion being masked by the thickened pleura. When pleural involvement is lacking the lung exhibits a localized or diffuse process suggesting abscess or tuberculosis. The mediastinum is not displaced and rarely exhibits involvement.

**Diagnosis**—The diagnosis is rarely made until pus is obtained from an abscess or sinus tract or by thoracentesis. Positive diagnosis depends upon identification of the specific fungus. If the visible sulphur granules are washed repeatedly with saline and the sediment macerated the fungus appears under the microscope as a dense central mass of mycelium with radiating filaments. The terminations of the filaments may or may not show budding. Tissue from a sinus may also reveal the organisms on section. Graham and his co-workers recommend the technique of Mandelbaum as given by Goldman.<sup>1</sup> The fluid is placed in an Erlenmeyer flask and allowed to stand overnight in an ice-box. The supernatant fluid is decanted and the sediment placed in a 50 cc. centrifuge tube and centrifuged for at least twenty minutes at moderate speed. The supernatant fluid is again decanted and the sediment hardened with a diluted solution of formaldehyde U. S. P. (1 to 10) or Zenker's fluid for twenty-four hours. The fixed sediment is then treated as ordinary tissue by running through alcohol imbedding in paraffin and staining with eosin hematoxylin.

Lord describes the various cultural characteristics of the fungus and the difficulties in differentiating it from sporotrichosis. He suggests the organisms may be related and that the latter may be a very virulent form although its clinical manifestations are different.

**Treatment**—Although there have been reports of cures by various methods none has proved to be uniformly satisfactory or even encouraging. The time-honored method of treatment has

<sup>1</sup> Goldman A. Arch. vet. of Surgery, December, 1929.

been the use of iodides in massive doses. Fortunately children tolerate potassium iodide in large amounts. Beginning with 5 grains three times daily, the dosage is rapidly increased to 60 or more grains daily. Ellis suggests that as an alternative, tincture of iodine be given in milk beginning with 2 minims and increasing the dose cautiously. Lugol's solution may be administered intravenously.

Roentgen ray therapy has been employed with variable results. Although the rays do not destroy the organism *in vitro* growth is inhibited. Those who advocate radiation recommend large doses every week or ten days over as many fields as necessary, care being taken to avoid damage to the skin.

Surgery has been tried with certain benefits. Wangensteen recently reported the successful use of multiple and extensive excisions of the diseased tissues. He recommends early surgical treatment consisting of radical excision of all infected tissue when possible, otherwise wide excision, incision and drainage.

It is possible that more favorable results will follow the early use of massive doses of iodides when combined with surgery or adequate radiation or both. Unfortunately many cases are symptomless until the disease has become extensive. Death usually occurs in six months to two years thereafter. Cases suffering from the aerobic type of the fungus offer a more hopeful prognosis than the anaerobic

### AMEBIOSIS OF THE LUNG

Amebiosis of the lung rarely occurs in children. In recent years, however, amebic infection has increased in the United States and it is probable that the disease will be found more frequently in young patients.

**Incidence**—Between 15 and 35 per cent of all amebiosis cases develop liver abscess and 5 to 15 per cent of the liver-abscess group have pulmonary complications. Ochsner and De Bakey collected 153 cases in the literature including 15 of their own in which pulmonary complications occurred. The youngest patient was fourteen years of age.

**Etiology**—The infecting organism is the *Endamoeba histolytica*. This occurs in two forms—the trophozoite, active or precystic and the cystic form. The active organism is 20 to 30 microns in diameter and is practically colorless with a fine uniformly granular endoplasm and clear ectoplasm. The nucleus is delicate, inconspicuous and often invisible. In stained specimens it is lined by fine granular chromatin. In the center of the nucleus the karyosome appears as a deep-staining mass surrounded by a clear zone. Between the latter and the nuclear wall there is an area of chromatin material. In the body fluids the ameba is actively motile and

appears elongated. After exposure outside the body motility ceases and its shape becomes rounded.

**Pathogenesis**—It is probable that amebic infection of the respiratory tract is never primary. The pulmonary complications occur as a result of hematogenous dissemination from the gastro intestinal tract or by direct extension from a liver abscess. Ochsner classified the pleuropulmonary complications in a collected series of 153 cases as follows:

- 1 Hematogenous pulmonary abscess without liver involvement 14.3 per cent
- 2 Hematogenous pulmonary abscess and independent liver abscess 10.4 per cent
- 3 Pulmonary abscess extending from liver abscess 37.2 per cent
- 4 Bronchohepatic fistula with little pulmonary involvement 19.6 per cent
- 5 Empyema extending from liver abscess 17.6 per cent

**Pathology**—Although the abscess may occur in any part of the lung, the most common location is in the right lower lobe. The well-demarcated fibrous capsule contains greenish yellow purulent material. Amebae are present in the pus and occasionally in the capsule. When secondary to liver abscess the contents consist of chocolate-colored viscid fluid.

**Symptomatology**—There is usually an antecedent history of diarrhea and pyrexia followed by right upper quadrant abdominal pain and at times severe right lower chest pain. Early in the disease there is a distressing non-productive cough which continues until evacuation of the abscess occurs. This event is characterized by the sudden expectoration of large amounts of pus or of chocolate sauce material.

The chest findings are confined to the area involved and are influenced by the amount of fluid in the cavity and the presence or absence of communication with a bronchus. The findings may accordingly be those of cavity or consolidation and if the pleural cavity is involved of effusion. Abdominal examination is important especially in cases secondary to hepatic abscess. In the latter the liver is enlarged and tender and there may be right upper quadrant rigidity. The temperature seldom exceeds  $101^{\circ}\text{F}$  except when secondary infection occurs.

**Laboratory Findings**—In any suspected case both fresh sputum and fresh stool should be examined for amebae on the warm stage. The organism is demonstrable in the sputum in only 10 to 30 per cent of the cases and in early types the findings are usually negative. The blood exhibits moderate leukocytosis with a relatively normal differential count. In Ochsner's collected series the total white cell counts were between 10,000 and 20,000 per cu mm with an

## CHAPTER XXX

### THE PLEURA

#### HYDROTHORAX

THE fluid producing hydrothorax is of non-inflammatory transudative origin. It is thin, clear, straw-colored, of specific gravity under 1.015, low in albumin content, free of cells and bacteria, and does not clot on standing.

**Etiology**—The transudate is secondary to some other disease, most frequently cardiac decompensation, nephritis with edema, nephrosis, intrathoracic tumor or leukemia. The fluid may develop in one or both pleural cavities and when unilateral is usually located on the right side due to pressure on the major azygos vein. The condition is often associated with ascites, hydropericardium or subcutaneous edema.

**Symptomatology**—A small amount of fluid is asymptomatic. Its presence may be discovered by physical examination, aspiration or roentgen-ray. Large accumulations produce mechanical interference with the circulation causing dyspnea and at times cyanosis.

**Treatment**—This should be directed toward the primary disease. Aspiration is indicated solely for diagnosis or the relief of pressure symptoms.

#### HEMOTHORAX

Frank blood in the pleural cavity occurs infrequently. The condition may follow chest trauma, aspiration, artificial pneumothorax, erosion of a vessel by a tumor or tuberculous focus, or the rupture of an emphysematous bleb at the periphery of the lung with concomitant tearing of a blood-vessel. Depending upon the etiology, there may be an escape of air producing hemopneumothorax, if a communication occurs with a bronchus, pyohemopneumothorax may develop.

**Symptomatology**—The amount and character of the hemorrhage determine the acuity and severity of the symptoms. The physical signs are those of either hydrothorax or hydropneumothorax. Diagnosis is made by the history, rapid accumulation of the fluid and the aspiration of blood.

**Treatment**—This should consist of absolute rest and, if necessary, blood transfusion. Aspiration should be done only if respiratory embarrassment is marked and threatens life, and then only in

amounts to afford symptomatic relief. Large withdrawals may prevent clotting at the site of bleeding and frequent tapplings may introduce infection. If the patient survives adhesive pleuritis follows. Calcification of the pleura may occur later.

Surgical intervention during active bleeding is hazardous as the patient is usually in poor operative condition. It may also be extremely difficult to locate the site of hemorrhage and the danger of infecting the cavity is obvious. Should infection develop, drainage becomes necessary.

### CHYLOTHORAX

The condition is rare, especially in children. The escape of chylous fluid from the thoracic duct or its branches is usually due to trauma and infrequently to pressure upon the duct by large nodes or tumors.

The milky fluid has a high fat content and often contains cholesterol, albumin, sugar and lymphocytes. It does not coagulate on standing. The fat seen as refractile microscopic globules can be further identified by staining with Sudan III.

**Symptomatology.** There is usually an interval of four to six days or longer between injury of the duct and the onset of symptoms. These comprise progressive cough, dyspnea, cyanosis and the signs of pleural effusion. Diagnosis rests upon aspiration of the milky fluid. At first this may be rose-tinted from admixture with blood.

**Prognosis.**—When the leakage is profuse, death results from depletion. The average mortality in traumatic cases is approximately 50 per cent.

**Treatment.** Chyle is essential for life and the problem is a most difficult one. Surgical intervention is impractical and futile. The customary treatment is repeated aspirations for respiratory embarrassment. In an endeavor to prevent the loss of chyle and aid healing of the duct, two procedures have been tried: (1) An attempt to reduce the formation of chyle through a fat-free diet or rectal feedings with nothing by mouth, and (2) thoriotomy to reduce the negative pressure which may act as a suction. Neither has proven successful.

Ocken (1905) administered the aspirated chyle intravenously but his patient died. Bruerfeld (1937) reports a cure following this intravenous method. The treatment is apparently without danger as the sterile chyle produces no reaction when introduced into the circulation. Infusions of 1000 cc. of chyle may be given following each aspiration.

## ACUTE EMPYEMA THORACIS

(*Purulent or Suppurative Pleurisy Pyothorax*)

Empyema thoracis is a collection of serum fibrin and pus cells in the pleural cavity. It is by far the most common thoracic pathology requiring surgery.

**Etiology** — The disease in almost all cases is preceded by or co-existent with broncho or lobar pneumonia more commonly the latter. Involvement of the pleura may occur by extension of the invading organisms through the lymphatics or by direct spread of the pneumonic process through the respiratory elements to the visceral pleura. Infants with sepsis frequently develop small abscesses of the lung which rupture into the pleural cavity. In such cases the organism is usually the hemolytic streptococcus. Ashby in a study of pneumonia and empyema in children found that 10 to 12 per cent exhibited peripheral abscesses. Putrid abscess of the lung and gangrene may also rupture into the pleural space.

Infrequent causes of empyema are subdiaphragmatic abscess, suppuration of the mediastinum or pericardium, puncture wounds of the chest, abscess in the soft parts, osteomyelitis of a rib and thoracentesis.

**Bacteriology** — Usually a single pyogenic organism is found in the pleural exudate. The most common in order of frequency are the pneumococcus, streptococcus and staphylococcus. Except in epidemics the usual invader is the pneumococcus and its particular type in large groups of cases may vary from year to year. Infrequent causative organisms are the tubercle bacillus, the anaerobes producing putrid abscess and gangrene of the lung, actinomycosis, streptothricosis and blastomycosis. In cases of draining chronic empyema or bronchopleural fistula there is frequently a mixed infection of two or more organisms. These secondary invaders make the treatment more difficult and less satisfactory.

**Pathology** — Pneumococcus empyema may develop any time during the course of pneumonia or shortly after the process has subsided. It is probable that more cases begin during the acute stage than are indicated by the clinical findings. Although at first the pus may be thin and clear it is usually cloudy with a yellowish cast. It thickens rapidly and contains stringy shreds of fibrin and assumes a greenish yellow color. The pus may become so viscid that it can be aspirated through the paracentesis needle only with difficulty. It consists of serum, fibrin, polymorphonuclear cells in various stages of degeneration and the invading organism. In rare instances the exudate becomes sterile.

Streptococcus pleural infections usually occur earlier than pneumococcus types and the amount of exudate is often larger. The fluid frequently appears serous at the onset and becomes frankly

purulent less rapidly than pneumococcus pus, also the fibrin content is less. Blood is present in variable amounts rather frequently. None of these features is characteristic, however, and the specific diagnosis depends upon identification of the organism.

*Staphylococcus pus* may resemble either of the aforementioned Empyema due to anaerobes or *B. coli* has a foul putrid odor. Whenever the pleural exudate is persistently serous or thin, with little fibrin, and especially if it is sterile when cultured on ordinary media, tuberculosis should be suspected and an attempt made to obtain the organism by special cultural methods and animal inoculation. Primary pleural effusions in which no etiologic organism can be determined should be considered tuberculous until proven otherwise. The fluid in tuberculous empyema may become serofibrinous, seropurulent or frankly purulent. The last, however, is rare in children. Tuberculous fluid is often distinguishable from other exudates by the predominance of lymphocytes.

The pleura in empyema becomes yellow, shaggy, thickened and even fibrous through the laying down of collagen fibers and the deposition of pus cells and fibrin. The rapidity with which this occurs influences the time when open evacuation of the pus may be performed safely. Thus, in streptococcus infections where the empyema is not collected in a localized area and where fixation of the mediastinum is slow, early thoracotomy may be followed by mediastinal flutter with its attendant burden upon the respiratory and circulatory systems.

Adhesions frequently form between the two pleural layers and the pus becomes encapsulated in one or several areas. The most common locations are laterally, posteriorly, and at the base; infrequent sites are near the apex of the lung and in the interlobar fissures. Localized empyemas are less serious than generalized types but exploratory paracentesis is more difficult unless the process is accurately localized by roentgen-ray or fluoroscopy.

Empyema may involve the pericardium and produce serous or purulent pericarditis, or the mediastinum with resulting localized or generalized mediastinitis. More frequently bronchopleural fistula, empyema necessitatis or pyopneumothorax develops. It is probable that most bronchopleural fistulae result from rupture of a pulmonary abscess into the pleural cavity rather than by invasion of the pleural infection into the lung substance. Irrespective of the mode of production air is introduced into the pleural space producing pyopneumothorax. The latter may also follow thoracocentesis through trauma to the lung or the ingress of air into the empyema space. Air is always present in open drainage cases and also at times when closed drainage is instituted. Gas bacillus infection is rare and when present will cause pyopneumothorax. Rupture of an empyema into or through the chest wall, termed empyema neces-



sitatis generally occurs at the thinnest part of the thoracic cage between the second and seventh ribs anteriorly.

In rare instances untreated empyemas become localized and remain so without producing complications and ultimately become absorbed. It is also probable that resolution occurs in certain cases in which the exudate is small in amount and not frankly purulent.

**Symptomatology** The most common sequence of events is seen in lobar pneumonia. The patient appears to have recovered from the primary infection as denoted by the drop in temperature to normal or almost normal, the remarkable decrease in toxemia and general improvement. The pulse however does not usually reach normal. After a period of one to several days the temperature rises and becomes septic in type with peaks reaching  $103^{\circ}$  to  $105^{\circ}$  F. Toxemia reappears and a dry hacking cough develops. Dyspnea may also occur if the exudate is abundant. In other cases especially those with streptococcus infection empyema develops before the pneumonic process has subsided and there is no particular symptomatology to indicate the subsidence of the primary disease or onset of the complication. The diagnosis under these circumstances depends upon the physical signs, roentgen rays or exploratory puncture. In certain instances there is a gradual but never complete subsidence of fever, cough and toxemia and the patients develop progressive anemia, loss of weight and weakness. Many are incorrectly considered unresolved or slowly resolving pneumonia.

The occurrence of a chill or sudden rise of temperature associated with collapse or sudden dyspnea should suggest pyopneumothorax. Expectoration of pus may or may not occur.

Severity of the cough and dyspnea depends chiefly upon the amount of fluid in the pleural cavity and the degree of toxemia. When a large exudate occurs without dehiscing pleural adhesions the mediastinum is displaced toward the contralateral side and marked respiratory and circulatory embarrassment may result. If the exudate is localized and no mediastinal displacement occurs a dry cough may be the outstanding symptom.

**Symptomatology of Chronic Empyema**—In chronic empyema the child usually presents the syndrome of chronic sepsis—low grade pyrexia and rapid pulse with progressive loss of weight and strength and secondary anemia. The involved chest frequently shows retraction with shifting of the mediastinum toward the diseased side. Clubbing of the fingers and toes may also occur. Although the physical signs are variable, dullness to flatness and diminished breath sounds are generally demonstrable. The presence of purulent sputum should suggest lung abscess with probable bronchiectasis or bronchopleural fistula with or without bronchiectasis. Any chronic draining sinus is also strongly suggestive of lung abscess or bronchiectasis or both. Other causes of chronic empyema are

osteomyelitis of the ribs inadequate drainage and failure of the collapsed lung to expand because of thickening and fibrosis of the visceral pleura

**Physical Findings**—The physical signs are essentially those of any effusion. The side of the chest involved usually shows limitation of motion and the interspaces are fuller than on the opposite side. Impulses of the heart and aorta are occasionally transmitted to the chest wall so that pulsations are seen. The apex beat may or may not be displaced toward the opposite side depending upon fixation of the mediastinum and the amount of fluid present. In old empyemas where there has been considerable contraction of fibrous tissue the cardiac impulse may be displaced toward the involved side. In such cases the diseased side appears smaller or flatter than the normal. Localized empyema frequently exhibits no cardiac displacement. Marked dullness or flatness associated with diminished or absent fremitus regardless of the type quality or intensity of breath sounds is presumptive evidence of fluid. The breath sounds may be entirely absent tubular or even amphoric and of loud intensity. The spoken voice may or may not be transmitted. Localized empyema may reveal only localized flatness without other abnormal findings. When the encysted process is on the diaphragm or in the interlobar fissures physical findings are generally wanting.

**Diagnosis**—With an antecedent history of pneumonia and persistence of fever or recurrence of fever within one to several days after an apparent crisis the diagnosis of empyema is probable. Persistent leukocytosis and rapid pulse are also suspicious signs. Roentgen ray examination of the chest usually verifies the diagnosis although thoracentesis may be necessary for confirmation. The roentgenographic and fluoroscopic pattern exhibit a uniformly dense shadow which becomes less dense toward the upper margin if the fluid is free in the pleural cavity when the fluid is basilar the upper margin is frequently curved with the highest portion in the axillary region. Encapsulated empyema appears as a fairly well circumscribed area frequently with a less dense periphery.

When the amount of fluid is massive the mediastinum is displaced to the opposite side the diaphragm is often depressed the interspaces widened and the involved thorax appears larger than its fellow. Frequently the spine can be seen to show curvature toward the normal side. Lateral and oblique views are often necessary for accurate localization. Interlobar mediastinal and diaphragmatic effusions are usually diagnosed only by roentgen ray and fluoroscopic examination.

*Thoracentesis is a necessary procedure before any therapy is considered. Examination of the aspirated fluid will reveal the specific organism and aid in determining the optimum time for surgical*

intervention. Although the exploring needle is indispensable in locating encysted collections of pus its injudicious use is to be condemned. If several attempts fail the area should be relocalized by roentgen ray and fluoroscopy.

**Thoracentesis Technic**—The child is seated and mummified. McEnery and Brennenman describe an excellent method of restraint. "An efficient method of restraining the arms is to pull the closed shirt up to the neck in back and over the arms in front and pin it securely so that the folded arms are held tightly in a kind of sling. The thighs are bound together with a towel or diaper. The assistant standing in front of and to one side of the patient passes one arm over the back of the child's neck and under the arms grasping the opposite arm, while with the other hand he grasps the farther thigh, holding the child firmly against his body. In this manner the head is flexed forward the scapula is elevated and the intercostal spaces are widened on the affected side while at the same time all undesirable motion is reduced to a minimum.

In large effusions thoracentesis is performed in the sixth seventh or eighth interspace in the posterior axillary line. Localized effusions are explored over the area determined by roentgen-ray and fluoroscopy, or the point of maximum dullness.

The area is sterilized with iodine and alcohol beginning at a point where the needle is to enter and cleansing peripherally. The skin, intercostal space and pleura are anesthetized with 1 per cent novocaine, using a No. 24 gauge needle 1 inch long. The interspace is entered by inserting the needle slightly above the upper border of the lower rib. When the fluid is very thin it may be aspirated with a hypodermic needle if only a diagnostic specimen is desired. Should the pus be thick or aspiration of large quantities be indicated a needle of No. 13 to 15 gauge 2 inches long and with a short bevel is inserted through the anesthetized area. The needle may be attached to a syringe and three-way stopcock, or to a piece of rubber tubing which is clamped the clamp being removed only after the pleural space has been entered, felt by a sudden 'give'. The tubing is attached to a satisfactory suction apparatus. Before removing the needle, a syringe containing a few cubic centimeters of 70 per cent alcohol should be attached to cleanse the needle while in place, as it is withdrawn small amounts of alcohol are injected to sterilize the puncture tract. An alcohol sponge is then strapped tightly over the puncture site.

Every effort should be made to prevent the entrance of air into the pleural cavity by maintaining a 'closed system' suction apparatus being certain that when using a Potain apparatus the connections are correct. The needle should not be introduced farther than a few millimeters beyond the parietal pleura and should not be moved about lest lung tissue be damaged. If the needle becomes

plugged by fibrin it may be reopened by injecting a small amount of saline

**Differential Diagnosis** —1 *Lobar pneumonia* may simulate effusion. In the former the onset is generally acute, the signs are less extensive and confined to one lobe, dulness is less intense, altered breath sounds are transmitted well riles are almost always present and the cardiac impulse is not shifted. *Tuberculous pneumonia* covering a large area may simulate effusion in all its findings except the change of cardiac outline. Repeated negative tuberculin tests exclude tuberculosis.

2 *Unresolved Pneumonia* —The temperature and toxemia are slight or absent and cough may or may not be present. The physical signs are those of pneumonitis with riles being the most outstanding finding. The usual signs of effusion are absent unless the pleura is considerably thickened. The roentgenogram does not exhibit a uniform dense even shadow but rather a mottled area confined to the site of the original pneumonia which shows gradual recession on progressive examinations. Depression of the diaphragm and mediastinal displacement are lacking.

3 *Serofibrinous Pleurisy* The onset may be insidious with few symptoms or acute with hyperpyrexia, chest pain and cough. There are no positive findings of pneumonitis and the toxemia is usually definitely less than in pneumonia or empyema. The disproportion between the constitutional symptoms and the physical findings suggest the diagnosis. When the effusion is massive cyanosis and dyspnea may occur. There is often a history of tuberculous contact and the tuberculin reaction may be quite severe. Aspiration of the fluid is indicated when the diagnosis is doubtful. Thin fluid with a predominance of lymphocytes is presumptive evidence of tuberculosis even though culture and animal inoculation fail to produce the organism.

4 *Lung Abscess* —There is little difficulty in differentiating the two conditions except when the abscess ruptures into the pleural space and produces a generalized or localized empyema.

5 *Actinomycosis* —The onset is insidious and frequently the patient is not seen until an actinomycotic abscess is about to rupture through the chest wall. Such dwellings are indurated, firm and brown. If aspiration is attempted the amount of pus withdrawn is usually disappointingly small. The ray fungus is readily demonstrable in the sulphur like granules. The sinns or sinuses which may form have no special characteristics but the discharge contains abundant fungi.

6 *Subdiaphragmatic Abscess* —This is rarely, if ever, secondary to pleural or pneumonic lesions. There is generally a history of antecedent abdominal infection especially appendiceal. (Refer to the section on Subdiaphragmatic Abscess.) The patient may

present a clinical picture of empyema and the subdiaphragmatic abscess may be overlooked at the time of thoracotomy. The presence of bile, *B. coli* or *typhæ* should suggest a subdiaphragmatic lesion.

**7 Atelectasis** —There is often a history of aspiration of a foreign body. The mediastinum is drawn toward the affected side, the diaphragm is elevated and the interspaces are narrowed.

**8 Fibrinous Adhesive Pleurisy** —The pleura may become thickened, scarred and adherent so that the pleural space becomes obliterated. The condition may result from chronic empyema which has undergone spontaneous healing. Although the signs and roentgen ray findings may simulate effusion, there is generally retraction of the chest wall and interspaces and displacement of the mediastinum toward the involved side. In doubtful cases thoracentesis should be performed. The writer has seen several cases where thoracentesis revealed only a very small amount of pus but this was sufficient to account for the patient's illness.

**Complications —Chronic Empyema** —This may be due to delayed treatment or inadequate drainage of an acute empyema, insufficient drainage from a lung abscess which has ruptured into the pleural cavity, presence of a bronchopleural fistula, osteomyelitis of a rib or failure of the lung to reexpand following drainage. The presence of a bronchopleural fistula can be visualized by the injection of lipiodol followed by fluoroscopy or roentgen ray. The injection of a dye such as methylene blue into the fistulous tract with subsequent appearance of the dye in the sputum also confirms the diagnosis.

**Empyema Necessitatis** —This may be mistaken for an abscess of the chest wall. The aspiration of pus and replacement by air will aid in establishing the diagnosis by roentgen ray. Simple incision of the abscess is seldom adequate for drainage of the empyema cavity. Where actinomycosis is suspected the pus should be aspirated and examined before attempting operation.

**Pyopneumothorax** —This has been previously discussed. The treatment is similar to that of pyothorax without air except when tension pyopneumothorax develops. This should be treated by continuous drainage under sterile water or an antiseptic solution. Rupture of a lung abscess is the most common cause of pyopneumothorax.

**Suppurative Pericarditis** is an uncommon complication and occurs more frequently with streptococcus than pneumococcus infections.

**Suppurative Mediastinitis** —This rare complication is probably due to suppuration of the mediastinal lymph nodes following invasion of the pulmonary lymphatics.

**Suppurative Peritonitis** —Infections of the peritoneal cavity may extend into the pleural cavity by way of the lymphatics or by rupture through the diaphragm; the reverse however rarely occurs.

and it may be assumed that the associated peritonitis is primary to the empyema. Cases of overwhelming infection in which both empyema and peritonitis develop are probably of bacteriemic origin.

*Osteomyelitis of a Rib* may result from resection of the rib or pressure necrosis due to firm tubing employed in closed drainage. The presence of a sequestrum will cause persistent discharge.

*Cellulitis of the Chest Wall* may follow thoracentesis or drainage. When an aspiration is completed the needle should be cleansed while *in situ* by injecting a small amount of alcohol and continuing the injection while the needle is withdrawn. Only slight pressure should be used in injecting the alcohol.

*Amylod Disease* develops chiefly in cases of chronic empyema. Patients exhibiting persistent albuminuria with or without hepatic or splenic enlargement should be investigated for amyloid disease.

**Prognosis in Empyema**—There are so many variable factors that it is impossible to draw any general conclusions from a single set of statistics. The mortality is high during the first years of life especially in young infants due to the incidence of complicating septicemia. After the third year the prognosis becomes progressively better the average death rate being 10 to 20 per cent. During the past decade the operative results have improved considerably. This can be accounted for in large measure by the realization that open operation in early empyema produces high mortality and that by waiting until the pus is thick the mediastinum fixed and the underlying pneumonia resolved the death rate can be definitely reduced. Repeated aspirations of course do not come under this category.

Variations in the predominant organisms and their relative virulence from year to year greatly influence the prognosis and the deaths from pneumonia roughly parallel those from empyema and its complications. During different years certain types of pneumococci predominate. In Type I infection the prognosis is good even without the use of serum. Very toxic cases however should be given the benefit of serum. Patients with demonstrable bacteriemia invariably have a poorer prognosis than those with sterile cultures. In general death is rarely due to the empyema itself but rather to the underlying pneumonia and such complications as sepsis, nephritis, mastoiditis, gastro-enteritis, the acute exanthems, failure to relieve respiratory stress in massive effusions and tension pyopneumothorax.

**General Treatment** Certain general measures are essential in the adequate treatment of any child with empyema. Absolute bed rest is imperative so long as constitutional symptoms are present. Nursing care which will make the patient more comfortable and provide the maximum of rest is always indicated. Sedatives may be employed provided they do not interfere with respiration or the

cough reflex. Of equal importance is the maintenance of a normal water-salt balance. Inadequate fluid intake should be corrected by the use of parental injection of fluids. Reported blood transfusions calculated on the basis of 20 cc per kilo of body weight up to 250 cc are extremely valuable. The nutritional state of the patient should be maintained when possible by means of a high caloric diet supplemented with sweetened drinks. Bell has shown that a negative nitrogen balance occurs in patients with this type of suppurative disease and the diet should accordingly contain more than the basal requirement of protein. Heliotherapy is definitely advantageous and the child should be out of doors in bed when feasible.

If spontaneous drainage has occurred the patient should assume a posture which promotes drainage. Sinus cases following empyema necessitatis should lie on the involved side; the dressings should be changed frequently and the skin kept as free from irritation and infection as possible. Cases which have ruptured into a bronchus obtain the best results by lying on the well side in slight Trendelenburg position. The patient often discovers the position which affords optimum drainage and comfort.

**Emergency Treatment**—Unless the amount of fluid or intrapleural tension is great enough to cause serious respiratory embarrassment emergency treatment is unnecessary. Patients with enormous effusions causing respiratory distress are best treated by aspiration. The amount of fluid withdrawn should be determined by the relief obtained. The appearance of pain, cough or bloody fluid is undesirable and is an indication to discontinue the aspiration.

When pyopneumothorax occurs and the intrapleural tension is sufficient to cause respiratory distress air and pus should be withdrawn as in cases with massive effusion. If the relief obtained is only temporary the probability is that a tension pneumothorax has occurred. In such cases especially when the empyema is acute and the mediastinum is mobile continuous drainage should be instituted. A dull needle is inserted into the pleural cavity and strapped to the chest wall. To the needle is attached a rubber tube whose opposite end is submerged just below the fluid level of an antiseptic solution contained in an open bottle placed on the floor. So long as bubbles of air are expelled under the fluid the pleural pressure is positive and drainage should be continued. When negative pressure occurs the fluid will rise in the tube and fluctuate with each inspiration and expiration. (Should the needle become obstructed fluctuations will cease.) When it is observed that the pleural pressure remains constant after the aspiration is discontinued for a reasonable length of time the needle may be removed. This may be determined by manometric readings or the tube may be clamped temporarily. If there is no respiratory embarrassment after sixty minutes the needle may be withdrawn.

**Surgical Treatment**—The purposes of surgical treatment are (1) To evacuate the pus (2) produce a sterile cavity (3) obtain complete expansion of the underlying lung so as to obliterate the empyema cavity and (4) maintain the patient in a good nutritional state. Procedures which produce the desired results in the shortest time with fewest complications, least deformity and lowest mortality are naturally the most ideal. In the methods to be discussed it must be realized that each case is an entity unto itself and the treatment must accordingly be individualized. The general condition of the patient, presence or absence of an underlying pneumonia, etiologic organism, duration of the disease, character of the pus, progress of the patient and the condition of the mediastinum will determine the type of therapy and the time it is to be instituted.

The principal methods of artificial drainage comprise (1) Repeated aspirations (2) closed drainage and (3) open drainage.

**Repeated Aspirations** McFerv and Brenneemann are responsible for the renewed interest in this mode of treatment which was first practised by Roe in 1844. In their experience with 168 cases, 114 were cured without operation. Dana Ochsner and Gage Block and Parrish and others have modified the procedure by replacing the evacuated pus with air. They report excellent results with this modification and reserve radical procedures for cases which fail to react favorably after repeated aspirations. The technic consists in slowly aspirating as much fluid as possible unless cough, pain or bloody sputum results. The procedure is repeated every two or three days or more often if necessary. When the reaction is favorable the aspirations are continued as long as pus is obtainable. McFerv and Brenneemann state that the presence of fibrin masses is not a valid objection since a sufficient quantity of pus can always be aspirated in the early stages and that later the fibrin becomes liquefied. They also believe it is unnecessary to aspirate all the pus. (It is probably impossible with any method to evacuate all the pus from the pleural cavity at one time.) Those who replace the aspirated pus by air contend that more complete evacuation can be obtained thereby, that trauma to the lung is minimized and that no great change occurs in intrapleural pressure.

Aspiration followed by the injection of such drugs as sodium taurocholate, optochin and other substances has been employed chiefly in European clinics. The results obtained are not impressive.

Although there have been many conflicting reports on the virtues of repeated aspiration, it is the only safe method to employ when the fluid is thin and the mediastinum is mobile. This applies to all early empyemas regardless of the etiologic organism. Moreover, the method should be continued if satisfactory progress obtains. When the results are not satisfactory, however, it cannot be emphasized too strongly that some other form of therapy should be



instituted provided the empyema is localized and the mediastinum is fixed

**Closed Drainage**—Because of the very high mortality of open drainage cases during the World War and the remarkable results obtained by closed drainage in a similar group by Deidrich this method became very popular and has remained so. (The use of continuous closed drainage was used as early as 1876 by Hewett and except for slight changes in technic the essential principles remain the same.)

The theoretical advantages of the closed method are that continuous drainage is obtained, a negative pressure allowing the lung to reexpand is maintained and air is not allowed to enter the pleural cavity, thus eliminating the chances of sudden pyopneumothorax. Practically, however, these advantages are not always secured. When the pus is relatively thin, continuous drainage is easily maintained but when it thickens and large fibrin masses form, as in pneumococcus infection, the tube often becomes obstructed and requires frequent cleansing. While it is true that a negative pressure is preserved, there is danger of tearing lung tissue and thus producing a bronchopleural fistula. Moreover, the empyema cavity does not become obliterated by the approximation of both pleurae through negative pressure but rather by the extension and traction of granulation tissue from original points of fusion of both pleurae. This occurs only after the cavity has become sterile. The third theoretical advantage of air not entering the pleural cavity is valueless with restless infants and young children. Furthermore, tissue necrosis gradually occurs about the drainage tube so that air does enter the pleural cavity.

A great disadvantage of closed drainage is that tubing of sufficient caliber to drain thick pus cannot be employed without danger of causing necrosis of the intercostal vessels or nerves. Moreover, the patient must remain in bed until the drainage is discontinued.

Continuous drainage has its critics and advocates and their reports are highly conflicting. Some advocate irrigations with various antiseptics such as Dakin's solution, others the tidal drainage of Hart, and still others various types of suction apparatus. There actually seems to be little if any advantage in closed drainage over that of repeated aspirations.

**Technic of Closed Drainage**—After preliminary sterilization of the field, the skin and pleura are anesthetized with 1 per cent novocaine solution. A small incision is made in the derma to allow for easier introduction of a cannula and trochar. The cannula should be of sufficient caliber to admit the largest catheter possible that will not cause pressure on the ribs. The free end of the catheter should be clamped so that no air will pass through it into the pleural cavity and the end to be placed in the chest should have multiple

fenestrations and be of the mushroom type. Although the tube should reach well into the dependent portion of the cavity, it should not be long enough to cause pressure upon the lung, occlude itself by kinking, or curl so that its end lies above the level of the empyema. When the trochar is withdrawn, the catheter is introduced through the cannula. The latter is then removed, leaving the catheter in place. After the patient is returned to bed, the free end of the catheter is connected to a tube which is placed under the fluid level of a bottle partially filled with water or antiseptic solution. It is safest to place a slit finger cot at the end of the tube. This acts as a valve in permitting free egress of pus and preventing ingress of air or fluid. When the system is completely arranged, the clamp occluding the catheter is removed.

*Some prefer that intermittent drainage be practised during the first twenty-four hours to obviate any sudden decompression of the lung.* When the pus thickens and the tube becomes plugged with fibrin masses, Dakin's solution may be used for irrigations to facilitate liquefaction of the fibrin and the washing out of pus. McLaehem advises irrigations with Dakin's solution every two to three hours, beginning the second postoperative day. These should never be given under great pressure and preferably from a container held not more than 18 inches above the level of the thoracotomy wound. Irrigations are definitely contraindicated in cases with broncho-pleural fistula.

When the drainage diminishes and the contents of the cavity show diminution by fluoroscopy, roentgen-ray and fluid capacity, the drainage tube should be partially withdrawn. Gradual shortening of the tube is continued until the empyema cavity is practically obliterated and sterile. Fever during the treatment indicates incomplete drainage, localized pocketing of pus, or some other complication.

**Open Drainage**—The failure of repeated aspirations or of the closed method to establish adequate drainage with consequent fall in temperature and general constitutional improvement is an absolute indication for open operation, provided the pus is thick and the mediastinum is fixed. It cannot be stressed too strongly that an open operation during the early stage of empyema, especially when there is an underlying pneumonia, is fraught with grave danger and high mortality.

The advantages of the open method are that an adequate space can be established for the drainage of thick pus and fibrin masses, convalescence is usually shortened, ambulatory treatment may be adopted at any time, and exploration can be performed when necessary. The objections to the procedure are that it may be shocking, especially to infants and very weak children, and that it leaves a scar. Neither of these is terrible if the child has failed to respond to either repeated aspirations or closed drainage.

Graham Ballou and Singer state that Open drainage after the pleural infection has become a true abscess offers the advantage of free and adequate drainage without the necessity of any complicated apparatus. They perform three or four aspirations during the first ten to fourteen days and if these fail to lower the fever by one or more degrees they presume there is probably an underlying pneumonia still present or an infection elsewhere. It is generally agreed that in *pneumococcus empyema* open drainage may be safely performed eighteen to twenty one days from the time of onset of the pneumonia. In cases of *streptococcus empyema* a longer period may be required before the fluid thickens and safe delimiting adhesions form.

The portion of rib to be removed depends upon the site of the empyema. It is imperative that the lowermost part of the cavity be drained regardless of its location. The usual site for resection is the seventh eighth or ninth rib in the postaxillary line. In resecting the ninth care must be taken not to injure the diaphragm.

In cooperative patients local anesthesia with 1 per cent novocaine may be employed. After the skin and subcutaneous tissues are infiltrated location of the empyema cavity is verified by introducing an aspirating needle. The periosteum overlying the portion of rib to be removed is then anesthetized also the intercostal muscles and their nerves. The periosteum is separated only from that portion of rib chosen for resection and not beyond so as to preserve blood supply to the remaining parts. About 2 cm of rib is generally removed and the ends may be protected with bone wax to prevent osteomyelitis. An incision is then made through the periosteal bed into the pleural cavity. Pus is promptly expelled as well as fibrin masses. Coughing which generally occurs aids in the extrusion. Occasionally manual removal of the fibrin is necessary. The drainage tube is then inserted. This should be of new rubber sufficiently stiff to prevent collapse and from  $\frac{1}{2}$  to  $\frac{3}{4}$  inch in diameter.

Carlson and Bowers employ a method of combined open and closed drainage by passing the drainage tube through a rubber sponge. After incision has been made into the periosteal bed the tube is inserted and the rubber sponge is strapped tightly over the wound with adhesive tape. After the patient is returned to bed the distal end of the drainage tube is connected with a tube under fluid as is done with closed drainage.

Irrigations with Dakin's solution may be necessary if large fibrin masses or thick pus clog the tubing. The occurrence of fever after drainage has been established should arouse suspicion of possible complications such as otitis media gastro-enteritis nephritis etc. If these can be excluded a pocket of pus should be sought. As the cavity becomes smaller the tube is shortened or replaced by one

of smaller caliber. This is seldom necessary before the eighth to twelfth day.

When the empyema cavity has become sterile and its content is so less the tube may be removed. The wound should be kept open however until the space is completely obliterated. The progress of all cases of empyema should be followed by fluoroscopy and roentgen ray.

**General Considerations** — The success or failure of surgical treatment depends upon sterilization of the empyema cavity and its ultimate complete obliteration. If these conditions are not obtained chronic draining empyema or a recurrence even years later is likely to occur. For this reason it is unsafe to allow the chest wound to heal until all evidence indicates that the cavity has become obliterated. Measurements of the fluid capacity of the cavity should be made daily and cultures should be taken frequently. Fluoroscopic examinations are indicated once a week and roentgen-ray studies every two weeks. The posture which exhibits the cavity best by fluoroscopy should be repeated when making the roentgen-ray studies. If necessary contrast substances such as lipiodol may be injected to determine the size and contour of the space.

It is probable that the various drugs and dyes employed for irrigation or installation help render the cavity sterile by removing pus, facilitating drainage and destroying the organisms. Nevertheless the defense mechanisms of the body play the dominant role in accomplishing this end and many cures are obtained without any such measures.

There is still considerable controversy regarding the advisability of irrigations, the solution to be used and the time it should be begun. The main objections are that toxic products are more readily absorbed thereby, bleeding may occur, pleural shock and even cerebral embolism may result and bronchial fistula may be produced. There is no actual evidence to prove that toxic absorption is enhanced. Bleeding occurs at times but is usually very slight. Upon discontinuing the irrigations for two or three days bleeding ceases and the irrigations may be resumed. Syncope during or following the treatments occurs very infrequently. Branchopleural fistula may develop but this cannot be attributed wholly to the irrigations. In certain streptococcus infections in which both the lung and pleura are involved fistulae can form easily. Their occurrence in children is usually of slight import. When present however the wound should be kept from closing and irrigations be omitted. The latter are extremely irritating to the bronchial mucosa and produce violent coughing. Following closure of the fistula irrigations may be resumed.

The most widely employed fluid is Dakin's solution. It should be freshly prepared and not over two days old. The solution

possesses antiseptic deodorizing and solvent properties. The last is probably its greatest attribute and fibrin deposits upon the pleura especially the visceral are removed rapidly. This is extremely important as the fibrin may otherwise become organized and prevent reexpansion of the compressed lung. Other solutions including saline and various dyes are occasionally employed. It is the writer's practice to use either Dakin's solution or azochloramide. In using the former the skin should be protected by vaseline dressings.

Thoracic surgeons are not agreed upon the time when irrigations should be begun. Whereas some institute the procedure immediately after surgical drainage repeating it every two to four hours others wait until twenty four hours after operation. Those who disfavor irrigations employ them only when the exudate is very thick and contains considerable fibrin. It is the practice of the writer to begin irrigations about twelve hours after operation and to repeat them every three or four hours. They are continued until the cavity becomes sterile and less than 10 cc in capacity.

After the patient has remained afebrile for a reasonable length of time and the disease has progressed favorably graded exercises and progressive activity are allowed. These aid more in recovery than the use of blow bottles or other devices designed to facilitate lung expansion.

### CHRONIC EMPYEMA THORACIS

The usual causes of chronic empyema are (1) Failure of the lung to reexpand because of the presence of a thick fibrous layer upon the visceral pleura or of fibrosis of the lung (2) failure to sterilize the empyema cavity and (3) presence of a foreign body. The effects of a thick fibrous covering have been discussed. Fibrosis of the lung may occur as a result of lung abscess or of chronic pneumonitis following the original infection. A persistent broncho-pleural fistula may also produce pulmonary fibrosis or prevent the empyema cavity from becoming sterile. Pocketing of pus occurs at times and may be responsible for the continuance or recurrence of the disease. Foreign bodies are a frequent cause of chronicity the commonest offenders being rubber tubing gauze pieces of rib or osteomyelitic sequestra.

While chronic empyema is not a common occurrence in children its presence should be investigated when an apparently cured patient continues to have slight pyrexia persistent leukocytosis cough or pain and fails to gain weight and strength. A persistent draining sinus should also suggest chronic empyema or osteomyelitis of a rib.

The empyema cavity may be investigated by fluoroscopy and roentgen ray. Radiopaque substances as iodized oils will reveal the size and contour of the cavity and the presence or absence of a

bronchopleural fistula. Direct visualization of the cavity may be made with the thoroscope. A simple method to determine the presence of bronchopleural fistula is to inject methylene blue into the sinus while the patient lies on the good side. He should remain in this position for several hours. If a fistula is present the dye will usually appear in the sputum.

**Treatment**—Operative treatment is always indicated in chronic empyema. The simplest procedure is to enlarge the sinus tract by the excision of tissue and the removal of regenerated bone. Sections of adjacent ribs may also require resection to permit complete exploration of the cavity. If multiple pockets or multilocular cavities are present the septa should be broken to form a single cavity. Foreign bodies are sought and removed if present. Large drainage tubes are then inserted and the cavity is irrigated with Dakin's solution or azochloramide. The wound should not be allowed to close rapidly. If these simple procedures are not effective more radical surgery such as a modified Schede operation may be required. Bronchopleural fistulae usually heal spontaneously. Cases which persist need sitate plastic repair.

### SPONTANEOUS NON-TUBERCULOUS PNEUMOTHORAX

The condition rarely occurs in children and the case reports are few in number. The causes noted comprise pneumonia, gangrene, emphysema, hydatid cyst, pertussis, congenital defects, lung abscess, and foreign bodies, also bronchiectasis, infarction and trauma to the pleural. Recent reports indicate that the majority of cases are due to lung abscess with rupture into the pleural cavity.

The pneumothorax is almost always unilateral and the degree of collapse is determined by the amount of air which enters the pleural space and the presence or absence of adhesions. In cases due to rupture of an abscess pyopneumothorax develops.

**Symptomatology** The symptoms are variable depending upon the amount of lung tissue collapsed and the mobility of the mediastinum. The author saw a case in an eight year-old child. Although there was 50 per cent collapse of one entire lung the only complaint was an unproductive cough for five days. In others there are sudden dyspnea and cyanosis. Cases of abscess, bronchiectasis or other degenerative diseases which suddenly enter shock should be suspected of pneumothorax.

The signs are usually classical: hyperresonance with diminished or absent fremitus and breath sounds. The latter are occasionally transmitted much better than one would expect with the degree of collapse seen in the roentgen ray. The mediastinum is usually shifted to the opposite side and considerable displacement may exist without causing untoward distress. The roentgenogram is

characteristic The collapsed lung shows a definite limiting line surrounded by a clear zone without lung markings The presence of fluid is indicated by a fluid level in the pleural space

**Treatment** — If the symptoms are not progressive and the patient is fairly comfortable it is best not to institute any therapy except sedatives to control the cough In most cases the air will be absorbed and any effusion which develops generally disappears rapidly Should the symptoms be severe or become progressive it may be necessary to aspirate air from the pleural cavity Before doing so the pleural readings should be recorded A negative pressure does not militate against the presence of pneumothorax because the child's mediastinum is quite mobile and positive pressure may not occur until there is marked displacement Two hundred or more cubic centimeters of air should be removed and the readings again recorded If the symptoms recur or the readings become less negative or positive within one half hour it is advisable to institute constant drainage by inserting a dull silver needle preferably with a slight curve just beyond the parietal pleura The needle is held in place by adhesive tape and its distal end is connected by rubber tubing to a water bottle containing antiseptic solution the tip of the tubing dipping a few centimeters into the fluid The bottle is placed about 18 inches below the level of the chest

As long as air escapes under the fluid its drainage should be continued When the fluid rises in the tube and remains there showing free fluctuations the patient may be given a trial without drainage for one-half hour by clamping the tube If there is no respiratory distress the needle may be withdrawn The use of oil of gomenal or concentrated glucose in the pleural cavity has been used in cases with persistent and recurrent pneumothorax It is not advisable to employ them when infection is present

## CHAPTER XXVI

### THE ESOPHAGUS

**Anatomy** —The esophagus begins at the lower border of the cricoid cartilage and ends at the cardiac opening of the stomach. In the new born the commencement and termination are opposite the fourth or fifth cervical and the ninth dorsal vertebræ respectively. In adolescents and adults the markings are one or two vertebræ lower.

In the neck the organ is in close relationship with the trachea, cervical spine, thyroid gland, common carotid artery, laryngeal nerves and thoracic duct, and in the thorax with the aortic arch, descending aorta, trachea, left bronchus, pericardium, pleura, both vagi, nerves and the diaphragm.

Its course is vertical and in the mid line except for antero-posterior deviations which conform with the spine and slight lateral curvatures to the left from the root of the neck to the fourth dorsal vertebra and at the diaphragm where it enters the esophageal foramen. These deviations are less pronounced in infants than in adults.

The length of the esophagus from the cricoid cartilage to the cardiac orifice is approximately 8 to 10 cm. at birth, 14 to 15 cm. at three years and 25 to 30 cm. after puberty. The distance from the teeth (or alveolar border) to the cardia is approximately as follows:

	Cm.
At birth	18
1 yr.	20
2 yrs.	23
5	26
10	28
15	33
Adult	40

The diameter of 5 mm. in the new born gradually increases as the child grows but wide variations occur between individuals of the same ages.

There are three normal constrictions: (1) The cricoid, at the beginning of the esophagus; (2) the aortic, where the aorta and left bronchus cross the organ; and (3) the diaphragmatic, where it passes through the diaphragm. The first and third are more marked than the second.

In early life the lumen of the tube is lined by a thin layer of stratified squamous epithelium. Although all the glandular elements are present, the number of deep glands is less than in the adult.



## MALFORMATIONS OF THE ESOPHAGUS

**Branchial Fistulæ and Cysts**—During fetal life the lowermost branchial cleft may open into the lower pharynx or upper esophagus. Failure of the cleft to close results in an external cervical fistula. The condition is generally unilateral and the fistula may communicate with the esophagus or end blindly. Should the tract become obstructed a cystic mass may develop which cannot be differentiated clinically from similar tumefactions of other origin. Simple fistulæ whether blind or communicating with the esophagus require no treatment. Cysts especially those which grow rapidly and interfere with respiration and deglutition should be excised early and completely.

**Diverticula of the Esophagus**—These are rarely if ever congenital and are practically always due to traction from adhesions of neighboring structures. Bronchial adenitis is the usual cause and the most frequent site of the diverticulum is at the level of the bifurcation of the trachea. The condition seldom produces symptoms and requires no treatment.

**Pulsion Diverticulum**—The pathology is actually a diverticulum of the pharynx being an acquired hernia of the mucosa between the circular and oblique fibers of the inferior constrictor muscle. Obstruction of food through failure of the cricopharyngeus muscle to relax produces the herniation. The patients usually experience difficulty in deglutition and often regurgitate food without any evidence of its having passed into the stomach. Fluids however are usually swallowed with ease. When the diverticulum becomes enlarged it may cause a visible swelling in the neck. The diagnosis can be made by fluoroscopy after a barium drink or the splashing noise obtained upon shaking the soft tissues. Endoscopy is the most accurate means of establishing the diagnosis.

**Treatment** Jackson has described a method for the removal of pulsion diverticula through the use of an endoscope to empty and transilluminate the herniation while an external surgical approach guided by the esophagoscope is made. Following dissection of the sac the esophagoscope is passed into the lumen of the organ and the neck of the sac is then ligated. Feedings are given through a catheter until healing has occurred.

The removal of the diverticulum is best performed by a two stage operation. The purpose of the first procedure is to free the diverticulum and seal it from the mediastinum to prevent mediastinitis. The sac is first aspirated and cleaned by lavage with a stomach tube. The latter may be left in place to aid the operator should difficulty be met in locating the herniation. The sac is approached through an incision along the anterior border of the sterno-cleido-mastoid muscle. The muscles and carotid sheath are retracted care

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In early life the lumen of the tube is lined by a thin layer of stratified squamous epithelium. Although all the glandular elements are present, the number of deep glands is less than in the adult.

- 3 Agnesia (no detectible remnant of a restricted interval)
- 4 Fistula communicating with the trachea or primary bronchus
  - (a) Esophagus otherwise normal
  - (b) Esophagus divided into upper and lower segments by regional stenosis atresia or agnesia the lower segment communicating with the trachea
- 5 Doubling in a short extent
- 6 Diverticula
- 7 Cysts



FIG. 154.—Congenital esophagotracheal fistula. Gastrostomy performed on second day. Death from bronchopneumonia.

**Treatment**—The surgical treatment of cases with tracheoesophageal fistula has been notoriously unsuccessful (Fig. 154). Where the obstruction is caused by a web in an otherwise normal esophagus the fold may be broken endoscopically with good results. Stenosis may be overcome by gradual dilatation with bougies. The procedure should always be done endoscopically and never blindly. Repeated treatments may be necessary for months and even years.

**Congenital Shortening of the Esophagus**—In this extremely rare anomaly the shortening of the esophagus may be such that a portion of the stomach lies in the thoracic cavity.

**Dilatation of the Esophagus**—This is always the result of some form of stenosis, the dilatation occurring above the site of obstruction. Treatment comprises relief of the stricture.

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- 4 Fistula communicating with the trachea or primary bronchus
  - (a) Esophagus otherwise normal
  - (b) Esophagus divided into upper and lower segments by regional stenosis, atresia or agenesis, the lower segment communicating with the trachea
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**Diagnosis.**—This may be made by the history and roentgen-ray visualization of a blind pouch following the intake of barium fluid. (Fig 154) If the upper segment communicates with the trachea, the mixture will pass through the tracheobronchial tree. If an attempt is made to pass an esophageal catheter, obstruction will be encountered 10 to 12 cm from the gum margin. Esophagoscopy will confirm the diagnosis.

**Prognosis**—The outlook is hopeless. Most infants die within a week of bronchopneumonia due to the aspiration of fluid into the bronchial tree.

**Treatment**—No satisfactory method has been found to carry on the nutritional demands of the infant. Rectal feedings and intravenous glucose have proven inadequate in promoting growth and nutrition.

Various surgical measures have been employed without success. Gastrostomy has proven ineffective as pneumonia practically always results from regurgitation of the stomach contents into the trachea. In an endeavor to prevent this, ligation of the esophagus at the cardia has been attempted prior to gastrostomy. This procedure is unsatisfactory as the ligature tends to cut through the esophagus. Moreover, secretions collect in the blind esophageal pouch and the infected content empties into the lungs.

In order to prevent the regurgitation of stomach contents, Levin has modified the gastrostomy as follows. Under local anesthesia an upper left rectus incision is carried downward from the rib margin. The stomach is retracted until the cardia is reached. The sub-diaphragmatic esophagus and the cardiac end of the stomach are then mobilized by blunt dissection. A rubber tube, passed under the esophagus, is used for traction. After 1 cm of the mediastinal esophagus has been pulled into the abdomen, the cardiac end of the stomach and the esophagus may be brought into the wound by traction on the tube. The peritoneum and rectus sheath are then brought together by mattress sutures under the cardiac end of the esophagus and stomach and a multiple purse-string gastrostomy is performed distal to the exteriorized portion. The upper and lower ends of the wound are closed with mattress sutures. A rubber catheter is placed under the exteriorized portion and fastened to the abdominal wall with adhesive tape.

The angulation thus formed at the cardia, proximal to the gastrostomy, prevents regurgitation when milk is given through the gastrostomy tube. Perforations which occur in the exteriorized portion of the stomach make it advisable to cut across it in two or three weeks and to reconstruct the gastrostomy.

The second stage of the operation consists of a cervical esophagostomy. At a future date, antethoracic esophagoplasty may be performed to establish continuity of the gastro-intestinal tract.

Jejunostomy has been found unsatisfactory.

Division and closure of the fistula at its junction with the trachea together with a connection of the esophageal segments by a tube or modified Murphy button has been suggested, also exteriorization of the lower segment making a dorsal esophago-gastrostomy for feeding purposes.

Lilienthal suggests a posterior mediastinotomy on the right side to locate the fistula if possible. If found it may be ligated through the esophageal tissue and the esophagus cut away leaving the tracheal fistula sealed by the ligature. The esophageal opening could be enlarged and connected with the upper segment by a rubber tube tied in place. The mediastinum may then be packed and a gastrostomy done. The danger of aspiration would be diminished and later the question of esophagoesophagostomy could be decided on. It is questionable whether such heroic procedures are ever warranted.

### CARDIOSPASM—ESOPHAGISMUS—SPASM OF THE ESOPHAGUS

The conditions of spasm generally develop during infancy and are definitely less common in older children. The patients exhibit difficulty in deglutition and often vomit immediately thereafter. The spasms may be intermittent or constant and are frequently accentuated by nervous excitement. Many patients take liquids well but have trouble in swallowing solid food. Some can swallow only warm or hot foods while others favor cold. The spasm disappears under anesthesia.

There may be considerable difficulty or even inability in passing a catheter past the spasmodic closure. This should be done with great care and preferably under vision. If food is given by gavage or during a period when the spasm is absent no vomiting occurs. The ultimate progress is usually good.

**Diagnosis.** This is made by the clinical history, particularly in hypertonic infants or neurotic children, and visualization of the barium feeding by fluoroscopy and roentgen ray. Endoscopic examination may be necessary to confirm the diagnosis and exclude the presence of any ulceration which may produce the spasm.

**Treatment.** It is interesting to note that following a barium meal and exposure to the roentgen ray for diagnosis the spasm often ceases. Whether this is due to the barium and its possible though unlikely dilating effect or exposure to the rays is unknown. Removal of the patient from his environment, intelligent nursing, correction of overanxiety on the part of the parents or of the child's psychic problems will often result in cure. Bromides and pheno-

barbital used in conjunction with atropine may be tried though their success is not striking. Cases which do not respond to medical treatment may require dilatation by endoscopic instrumentation.

### CATARRHAL AND FOLLICULAR ESOPHAGITIS

Acute catarrhal esophagitis may occur at any age. It is usually secondary to acute infectious diseases, catarrhal inflammatory processes elsewhere in the gastrointestinal tract or to trauma produced by foreign bodies or hot liquids. The edema and injection of the mucous membrane and underlying connective tissue may be accompanied by superficial ulcerations. The condition is seldom severe and may be asymptomatic or attended by slight pain on swallowing. Cool bland drinks cause the least discomfort.

Esophagitis may also occur secondarily to diphtheria in which a typical diphtheritic membrane is found. Thrush, variola, pseudo-membranous infections, syphilis and involvement of adjacent caseating lymph nodes are rare etiologic factors.

Chronic catarrhal esophagitis is very uncommon in children. It may be a sequel of acute catarrhal involvement or be secondary to chronic cardiac or pulmonary disease.

Follicular esophagitis is characterized by enlargement of the mucous follicles which may exhibit superficial ulcerations. The condition is much less common than the catarrhal type and is usually found in association with other diseases of the digestive or respiratory tracts.

### ACQUIRED STENOSIS—STRICTURE OF THE ESOPHAGUS

The most common cause of stricture of the esophagus in children is the swallowing of caustics or strong acids. The pathology may vary from superficial necrosis to complete destruction of the mucous membrane. In severe cases the mucous membrane sloughs and is replaced by fibrous tissue which contracts and produces stenosis. Trauma caused by the swallowing of a foreign body or its removal may also produce stricture. Diseases such as diphtheria, syphilis, variola, caseation of adjacent lymph nodes, mediastinal abscess and tumors are rare causative agents. The type following corrosive esophagitis is usually located in the upper third of the organ. It may, however, occur at any zone and the stenosis may be annular or cylindrical. The esophagus becomes dilated above the obstruction and food may rest in the distended portion for hours after ingestion.

**Symptomatology**—Although stenosis frequently follows corrosive esophagitis, symptoms thereof may not appear until weeks or even months after the acute process has subsided. The children first

develop difficulty in swallowing solids. They masticate their food an unusually long time to make it as fluid as possible and frequently show evidence of dysphagia after it has been swallowed. Regurgitation often occurs and in severe cases even liquids cannot be swallowed. Marked emaciation follows unless means are provided to supply adequate nutrition.

**Diagnosis**—With a history of swallowing a corrosive, followed by dysphagia and regurgitation, the diagnosis is made easily. In cases less obvious it is noticed that there is progressive dysphagia especially of solids and regurgitation of food which shows no gastric digestion. Barium feeding visualized fluoroscopically and by roentgen ray and endoscopic examination, confirms the diagnosis. Blind passage of a catheter is a dangerous procedure.

**Treatment**—In corrosive esophagitis surgical treatment should not be instituted until three or four weeks have elapsed and the acute symptoms have subsided. The procedure in almost all cases comprises gradual dilatation of the stricture by bougies under endoscopic vision. The endoscopic treatments may be required for months or even years to maintain adequate dilatation. In cases in which it is impossible to pass even the smallest bougie, gastrostomy should be performed. Retrograde dilatation through the cardia is at times feasible.

In gastrostomy patients every effort should be made to maintain nutrition through adequate intake of milk, fruit juices, cream, eggs, olive oil, powdered vegetables and predigested starch foods. It is important that sufficient amounts of vitamins and minerals be incorporated in the diet.

Most cases which survive the acute esophagitis and maintain a satisfactory nutritional state will respond to endoscopic dilatations. Only the most severe types require gastrostomy. Plastic operations for restoration of the esophageal lumen are ill advised in children.

### REMOVAL OF FOREIGN BODIES

With the present methods of removal of foreign bodies by means of the esophagoscope it is rarely necessary to perform any surgical operation upon the esophagus. However, if the foreign body is so impacted in the cervical region that its removal by endoscopic means is impossible or unsafe, external esophagotomy should be performed. Approach is made through an incision on the left side of the neck along the anterior border of the sterno-cleido-mastoid. The muscles and carotid sheath are retracted and the esophagus brought into view. The foreign body is removed through a longitudinal incision in the organ. The wound is then meticulously closed with a double row of sutures and ample drainage is instituted to prevent mediastin



itis. Development of the latter is often fatal. Foreign bodies located near the cardia which cannot be extracted endoscopically may be removed from below through a gastrotomy approach.

### RETROESOPHAGEAL ABSCESSSES

Retroesophageal abscess occurs very rarely. The usual causes are the same as those of retropharyngeal abscess: also pleuritis, pericarditis, ulceration by a foreign body in the esophagus, ulceration by a tracheotomy tube, suppurative mediastinal lymphadenitis and spinal caries. The abscess forms behind and around the esophagus.

**Symptomatology**—There is no characteristic symptomatology. In addition to fever and dysphagia, dyspnea and cough may be present. The diagnosis is made by the history, distortion of the esophagus and presence of mediastinal swelling exhibited by roentgen-ray.

The abscess may rupture into the esophagus and heal spontaneously. More frequently, however, rupture occurs into the trachea, bronchus or lung producing purulent bronchopneumonia. Death may also occur from pressure on the vagi or from asphyxia through compression of the trachea. When recognized, the abscess may be incised and drained through the esophagus by means of the esophagoscope. The prognosis, however, is poor.

## PART VII

# THE ABDOMEN.

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### CHAPTER XXVII

#### THE ABDOMINAL WALL

##### APPLIED ANATOMY OF THE ABDOMINAL WALL

DUE to the transverse arrangement of the Langer lines of skin cleavage vertical scars tend to widen whereas horizontal ones generally produce a casual scarless scar

**Muscles**—The muscles of surgical importance comprise three groups (1) Anterior consisting of the recti and pyiformis (2) Lateral the external and internal oblique and transversalis and (3) Posterior the psoas quadratus lumborum latissimus dorsi and lumbar fascia The strong rectus sheath completely envelopes the muscle in its upper three-fourths below the semilunar fold of Douglas the posterior sheath is wanting

**Blood Supply**—This is mainly from the superior and inferior deep epigastric arteries also the musculophrenic superficial epigastric superficial iliac and external pudic

**Nerves**—The innervation of the abdominal wall is chiefly from the lower six intercostal nerves They are so distributed that the skin and parietal peritoneum are the more sensitive layers After piercing the base of the diaphragm and the transversalis muscle the seventh to tenth dorsal nerves pass forward between the internal and external oblique muscles to the posterior rectus sheath and thence through the muscle and anterior sheath to supply the skin area from the umbilicus to just below the navel The eleventh and twelfth nerves pursue a similar course and supply the lower abdominal wall to the pubis

After penetrating the psoas muscle and running forward on the surface of the quadratus lumborum the iliohypogastric nerve passes forward between the internal oblique and transversalis muscles to within 1 to 2 cm internal to the anterior superior spine It then passes through the internal oblique to continue its course between the two oblique muscles finally piercing the external to become subcutaneous above and to the outer side of the external inguinal ring The ilioinguinal nerve pursues a parallel course 1 cm below

the iliohypogastric and passing through the inguinal canal with the spermatic cord emerges through the external ring to become subcutaneous. The genitocrural nerve upon reaching the inguinal ligament sends one branch through the inguinal canal to supply the scrotum and adjacent part of the thigh and a second to the zone of the femoral triangle.

**Pain**—The spinal nerves communicate with the sympathetic (splanchnic) by means of the gray rami (visceral) and the white rami (vasomotor). The parietal peritoneum is extremely sensitive to pain and when irritated produces reflex spasm of the overlying muscles. Whereas organs which have only a sympathetic innervation such as the visceral peritoneum, omentum, visceral mucosa, liver, gall bladder, spleen, pancreas and kidneys do not register either pain or tactile sensation, the neck of the gall bladder, the biliary ducts and the ureters are extremely sensitive to pain. The mesentery has non-localizing sensation leading to epigastric distress and nausea; traction upon it however may produce colic.

Visceral reflex arcs carry impulses from irritated abdominal viscera through the splanchnic gray rami and lower intercostal or lumbar nerves to the overlying parietal muscles; the resulting spasm of the latter produces involuntary rigidity of the abdominal wall. The area of muscle spasm usually lies directly over the focal pathology. *In infants however hyperesthesia of the skin is often more pronounced than muscular rigidity.*

**Peristalsis**—The vagus is the motor nerve of the stomach and intestines; peristaltic movements in the latter are initiated by the intrinsic ganglia of Auerbach and Meisner. The vasomotor nerves inhibit peristalsis.

**Incisions**—The abdominal wall in infants is thin, especially in the mid line, and vertical incisions through the linea alba should be avoided. A paramedian approach 1 to 1.5 cm. laterad is definitely preferable. After the anterior rectus sheath is opened, the muscle is separated from the linea alba and gently retracted outward; the posterior sheath with the transversalis fascia and peritoneum is then elevated between artery forceps and opened carefully during inspiration. In closing wounds the peritoneal edges should be meticulously approximated with No. 0 or No. 1 plain catgut; otherwise a tab of omentum may herniate and favor wound gaping. The anterior sheath is closed with No. 1 chromic retention sutures of silk worm or dermal; are also advisable. Infants and young children are especially subject to dehiscence and it is best to support the wound for at least two weeks by broad adhesive straps covered with a muslin binder.

Some surgeons prefer a transrectus incision, splitting the muscle at the junction of its middle and outer thirds or temporarily dislocating it inward after the anterior sheath has been divided (Ham

merer) Both methods injure the nerve supply, damage to the deep epigastric veins may also cause troublesome bleeding. The advantage of the transrectus approach is that it is rapid and well suited to elongation for exploration.

The McBurney (gridiroo) incision is a muscle-splitting technic. The skin is incised parallel to Poupart's ligament (or transversely), the external oblique aponeurosis is divided obliquely in the direction of its fibers and the internal oblique and transversalis muscles are bluntly divided transversely, the transversalis fascia and peritoneum are then opened transversely. It is ill suited for enlargement or adequate exploration, also in purulent pathologies requiring drainage. The incidence of postoperative hernia is higher than in the transrectus approach. The incision has lost much of its former popularity.

### THE UMBILICUS

During early fetal development the lower ileum and cecum are contained within the umbilical cord outside of the abdominal cavity, being later withdrawn therefrom. The peritoneal covering extending into the cord is also gradually retracted and at birth presents as a slight depression at the umbilical ring. Defective intra-abdominal retraction of either the intestine or peritoneum eventuates in hernia into the cord.

Following ligation of the cord the umbilical ring closes as follows. The stump, covered by amnion, dries and sloughs off and the granulating area becomes epithelialized, the superficial fascia transversalis fascia and peritoneum fuse with the skin and produce the thin cicatrix which normally covers the umbilical ring, also the rectus sheaths do not quite come into contact. Fibrous obliteration of the hypogastric arteries and urachus strengthens the lower half of the ring and tends to pucker the umbilicus inward, whereas obliteration of the large umbilical vein leaves a definite weakened area in the upper portion. It is through this hiatus formerly occupied by the umbilical vein, that infantile and adult types of umbilical hernia evaginate, the rounded protrusion appearing above the cicatricial depression. (The varieties of exomphalos and their treatment are discussed in the section on Umbilical Hernia.)

**Anomalies** — Anomalies of the omphalo-mesenteric duct or of the urachus may result in fistula, cyst or diverticulum formation. Those of the former are discussed under "Meckel's Diverticulum."

The urachus, connecting the urinary bladder with the allantois normally becomes obliterated during the fifth month of fetal life and forms the vesico-umbilical ligament. Its persistence may be evidenced at birth by the presence of an umbilical sinus or fistula or the condition may develop after months or years. The discharge may be mucus, pus or urine.

**Treatment** — In similar cases with absence of urinary discharge an attempt may be made to obliterate the tract by the injection of such escharotics as Cutler's solution. A urinary fistula however should never be injected with caustics its treatment comprises excision after the cystitis has been relieved.

**Omphalitis in the New born** — The umbilical cord separates normally about the fifth day and epithelization is complete by the twelfth to fifteenth. Infections delay healing and the stump may become excoriated and exude pus occasionally true ulceration develops and rarely gangrene.

**Treatment** Prophylaxis comprises aseptic obstetrical technique and drying of the cord with antiseptic dusting powders such as boric acid, salicylic acid and starch, iodoform or thymol iodide. In the presence of mild infection 1 per cent silver nitrate may be applied daily. Cultures should always be taken for occasionally tetanus or diphtheria develops in the stump. In such cases 1000 units of the specific antitoxin should be injected subcutaneously. In severe infections with redness and infiltration of the surrounding tissues warm wet antiseptic dressings are indicated. Abscesses require incision and drainage.

Gangrene is a rare complication which occurs chiefly in premature infants from filth. As the process develops a greenish black area appears surrounded by a red areola. When the slough involves a considerable portion of the abdominal wall fatal peritonitis usually results or rarely a fecal fistula. Treatment consists in the application of continuous warm antiseptic dressings. Sterilization may be attempted with the endotherm coagulation current. Repeated small blood transfusions are supportive.

**Omphalitis in Older Children** — Dirt and sebaceous material often collect in the umbilicus of children. Erythema, ulceration or granuloma may result therefrom and rarely inspissated material forms a concretion (*cholesteroloma*). Chronic inflammation is generally due to dirt, eczema, syphilis or tuberculosis.

**Treatment** Cleanliness cures most cases. At times it is necessary to dilate or divide the umbilical ring in order to keep the parts clean. Antiseptic dusting powders such as boric acid or thymol iodide may be applied after bathing. Indolent granulations should be cauterized with 1 per cent silver nitrate. Tuberculosis and syphilis require systemic treatment.

**Umbilical Hemorrhage** — Hemorrhage from the navel may be accidental or idiopathic. In the former bleeding occurs from the umbilical vessels and in the latter from the umbilical tissues.

Accidental Hemorrhage may follow improper ligation, avulsion or premature separation of the cord. This type of bleeding is extremely serious and the infant may become exsanguinated rapidly. The

blood volume at birth is only one-nineteenth of the body weight (250 to 300 cc) and the loss of a few munces is often fatal

*Treatment* —Whereas oozing may be controlled by the application of styptics and pressure compresses frank hemorrhage requires re-ligation. A short stump may be ligated by first transfixing it with two straight needles inserted at right angles to each other and tying the ligature beneath them. Transfusion is urgently indicated if the loss of blood has been excessive

**Idiopathic Hemorrhage** is a variety in which spontaneous bleeding occurs from the umbilical tissues. The causes are manifold and but little understood. Trauma, prematurity, sepsis and congenital syphilis appear to be factors. The bleeding usually occurs after the cord has separated and the condition is often associated with hemorrhage in other parts of the body: liver, spleen, kidneys, peritoneum, spinal cord, nose and skin.

*Treatment* —The cause should be treated if determinable. Local measures comprise the application of firm dry dressings with or without astringents. Adrenalin locally is of questionable value. The administration of calcium is based upon a false hypothesis that the blood calcium content of the new born is low; it is actually relatively high. Specific treatment comprises the injection of whole blood either intravenously or intramuscularly; intraperitoneal injection is ill advised. Should a donor be unavailable horse or rabbit serum may be substituted.

**Tumors of the Umbilicus** —These comprise the following: warts, papilloma, fibroma, angioma, sebaceous and dermoid cysts. At times the tumors are congenital. Cysts occasionally develop to a considerable size and become infected. Warts, papilloma and angioma may be treated by fulguration; cysts and fibroma should be excised.

## THE PERITONEUM

The peritoneum exercises a vital role in health and disease. Composed of flat endothelial like polygonal cells on a thin basement membrane it lines the abdominal cavity and intimately invests the contained viscera. It is closely associated with an underlying layer of strong fascia except at certain places where fat and connective tissue occur.

**Blood and Lymph vessels** —The blood supply is derived from the abundant vascularity of the abdominal wall and viscera. Venous return occurs chiefly through the inferior vena cava and to a less extent the portal system. The rich lymphatic network drains directly into the abdominal lymph glands and through the lymph channels of the diaphragm into the mediastinal nodes. The thoracic duct is essentially a collecting vessel for the lacteals and its drainage of lymph is negligible.

**Nerves** —The parietal peritoneum is innervated through the lower intercostal and lumbar nerves whereas the peritoneum covering the central portion of the diaphragm is supplied by the phrenic and the peripheral part by the intercostals. Irritation of these respective peritoneal areas produces pain that of the parietal peritoneum is referred to the overlying abdominal wall the central diaphragmatic area to the neck and shoulders along the trapezius muscle and the peripheral portion to the lower costal and upper abdominal areas. Diaphragmatic pleurisy may thus simulate an intra abdominal lesion. The visceral peritoneum is innervated through the sympathetic system and is insensible to such stimuli as cutting or burning. Inflammation however produces pain likewise traction upon the mesentery. Apparently the sensory fibers which register pain accompany the sympathetic.

The peritoneal cavity may be considered a potential space containing but a few cubic centimeters of thin serum which permits visceral movement without friction. Closed in the male the cavity communicates externally in the female through the Fallopian tubes uterus and vagina (a potential source of infection).

**Spaces and Fossæ** Certain spaces and fossæ of the peritoneum are concerned respectively with the localization of suppurative processes and the development of internal herniæ. The Subphrenic space above the transverse mesocolon containing the liver stomach duodenum spleen and pancreas is divided into a right and left half by the falciform ligament. Purulent exudates may accumulate between the diaphragm and liver (subdiaphragmatic abscess) or beneath the liver (subhepatic abscess). The Lesser Peritoneal Cavity a huge fossa between the stomach and posterior parietal peritoneum communicates with the greater cavity through the foramen of Winslow. The cavity becomes involved in diffuse general peritonitis and in rare instances loops of small intestine may invaginate through the foramen and become strangulated.

The Lumbar Gutters or lateral fossæ of the mid abdomen contain the ascending and descending colon and function as dependent reservoirs for the collection of peritoneal exudate. The Pelvic or lower abdominal cavity is small in infants and young children and bacterial invasion readily provokes sepsis and irritability.

Peritoneal recesses may also occur in the duodeno-jejunal ileocecal and sigmoidal regions. Loops of gut occasionally invaginate therein and produce internal herniæ. (Refer to Internal Hernia.)

**Congenital Malformations** —Rotation of the gut may be interrupted at any point and thereby produce peritoneal malformation. Peritoneal bands or folds or fossæ may also be over or under developed. The chief congenital membranes of surgical interest are (1) Lane's kink a band stretching between the terminal ileum and pelvic peritoneum and (2) Jackson's membrane spreading out

from the parietal peritoneum to the ascending colon and often adherent to the omentum. Unless the membranes produce definite symptoms they should not be disturbed.

## THE MESENTERY

**Abnormalities** —The mesentery of the small intestine may be abnormally lengthened or shortened whereas that of the large bowel is subject to numerous abnormalities due to incomplete peritoneal fusion or arrested rotation. A long mesentery to the cecum produces cecum mobile. The ascending colon may exhibit a free mesentery. The normally short mesentery of the descending bowel may be greatly lengthened and in rare instances the small and large intestine may have a free common mesentery.

**Injuries** —The mesenteric blood vessels, lymphatics and lacteals may be damaged through operative trauma or crushing injuries. Many of the arteries are terminal vessels and the division of a main branch such as the ileocolic in appendectomy may lead to intestinal infarct and resulting gangrene. Extensive traumatic thrombosis may also result in devitalization of the gut and the damage of smaller vessels may cause mesenteric hematoma or hemoperitoneum. Although the intra abdominal hemorrhage in severe injuries may be due solely to mesenteric bleeding in most instances other viscera are involved especially the liver or spleen.

**Thrombosis** —Mesenteric thrombosis may result from trauma, infection, strangulation or volvulus. (Refer to Appendicitis, Strangulated Hernia and Volvulus.) The gut becomes edematous, dark in color and filled with blood and the latter may appear in the stools. Unless the circulation is reestablished gangrene and peritonitis eventuate.

**Mesenteritis** —Inflammation of the mesentery accompanies intraperitoneal infections. In chronic mesenteritis (tuberculous) the mesentery becomes thickened and shortened and the enlarged lymph nodes may fibrose, calcify or caseate.

**Tumors and Cysts of the Mesentery** —Primary tumors either lymphoblastoma or sarcoma are rare. Cysts are more common. Tving classifies them as follows: (a) *Chylous Cysts*. These are either dilated lymphatics or lymphangiomas. They may occur in the mesentery or omentum and contain clear fluid or chyle. (b) *Enteric Cysts*. These may arise in the mesentery or in the intestinal wall also in Meckel's diverticulum, persistence of the omphalo-mesenteric duct or from displaced portions of intestine. They are lined with epithelium and contain mucus. (c) *Dermoid Cysts*. The cysts occur rarely, are lined with epithelium and contain hair and sebaceous material. (d) *Nephrogenic Cysts*. Originating in the



retroperitoneal space, probably from remains of the Wolffian body, the cysts develop slowly and project into the peritoneal cavity.

**Symptomatology**—Cysts may be asymptomatic or produce discomfort through tugging upon the mesentery. In rare instances they may cause volvulus. The tumefactions are non-tender and are movable laterally.

**Treatment.**—Chylous and dermoid cysts should be enucleated cautiously in order to avoid damage to the mesenteric vessels. Serous and hemorrhagic cysts may be aspirated. Enteric cysts often require intestinal resection.

### THE OMENTUM

The omentum is an excessive redundancy to the left and downward of the dorsal mesogastrium. The anterior and posterior lamellæ, at first composed of two layers each, fuse early so that the lesser sac is limited by the curvature of the stomach and the posterior layers are fused to the transverse mesocolon.

The structure is highly vascular and contains numerous cell nests, the progenitors of wandering phagocytes which appear in peritoneal irritation. Its movement is totally extrinsic, respiratory excursion, peristalsis and posture being the factors which influence its change of position. Rapid adhesion to inflammatory foci and foreign bodies results from its excessive exudation of fibrin.

**Abnormalities.**—The omentum may be overdeveloped, aplastic, or absent, it may also exhibit clefts, fenestra, fusional failure of its leaves, or accessory omenta. All of the foregoing are rare. The length and fat content are normally quite variable and in early childhood the short frail omentum offers but slight protection in delimiting peritoneal infections.

**Injuries.**—Omental bleeding exhibits little tendency to spontaneous hemostasis and crushing or tearing injuries are accordingly dangerous from the standpoint of hemorrhage. In suturing rents in the omentum, care must be exercised not to perforate any blood-vessels as an extensive hematoma occurring between its leaves may lead to infection or cyst formation. In the absence of infection, disconnected portions of the omentum become revascularized through peritoneal adhesions and survive; in a septic field, however, they become necrotic.

**Omentitis.**—Inflammation of the omentum commonly accompanies bacterial invasion of the peritoneal cavity. The process is highly protective and tends to delimit the infection. Omentitis may also follow operative trauma, particularly if the structure has been ligated with silk, and in rare instances it may result from metastatic or embolic origin. **Torsion of the omentum** rarely develops in childhood and is usually associated with hernia. The symptoms

of acute abdominal pain nausea vomiting tenderness and muscle spasm mimic those of strangulated hernia or appendicitis. Although spontaneous cure may follow exploration is safer for the torsioned segment may become gangrenous. Chronic omentitis occurs commonly in tuberculous peritonitis the omentum becomes shortened thickened and matted into a firm mass.

**Cysts**—Very infrequently cysts develop from hemorrhage lymphatic block or necrosis. Tetral inclusions (dermoid and teratomas) and cystic lymphangiomas are exceedingly rare.

**Tumors**—Solid tumors arising between the leaves of the omentum are rare. They comprise lipoma fibroma and sarcoma.

**Omentula (Appendices Epiploicae)**—Omental tabs are subject to inflammation adhesion torsion and gangrene. Adherence to a loop of adjacent gut may produce intestinal obstruction or cause detachment of the omentula. Lying free in the abdominal cavity the tab may become cystic or calcified. Torsion of an appendix epiploica may eventuate in hemorrhage chronic inflammation fat necrosis or gangrene.

### INTRA-ABDOMINAL INJURIES

Severe intra abdominal injuries may result from either crushing or penetrating trauma. In the former variety blows kicks and especially run over accidents are common causes in the latter stab or bullet wounds.

Although crushing of the abdomen without evidence of intra abdominal damage is capable of producing shock in the great majority of instances the condition is associated with intra abdominal bleeding or the rupture of a solid or hollow viscus or both. Such cases often present a grave diagnostic problem. The salient differential symptomatology between perforation of a hollow viscus and that of hemorrhage is frequently clouded by the element of shock. In the final analysis the important factor is to determine whether or not an intra abdominal lesion has occurred which demands immediate surgical intervention.

**Shock and Hemorrhage**—Much can be determined by careful examination. In shock the child is pale with moistened brow cold extremities and lies quietly the temperature is subnormal the pulse rapid and of low pressure and the quantitative and differential white cell counts remain normal. In the presence of hemorrhage restlessness and especially thirst are evidenced the pulse rate rises progressively and leukocytosis is present.

**Intra abdominal Bleeding**—In profuse intra abdominal bleeding the entire abdomen may be tender and there may be flank dullness on one or both sides. In run-over cases a wheel mark across the right or left upper abdomen suggests the probability of rupture of

the liver or spleen respectively and in the mid or lower abdomen, rupture of the mesenteric vessels or small intestine

Rigidity of the abdominal muscles occurs in both hemorrhage and intestinal perforation. When localized at the site of injury it is often difficult to differentiate reflex involuntary rigidity from voluntary spasm due to intramural hemorrhage. With perforation of the intestine a crescentic subdiaphragmatic air bubble is often demonstrable in a flat roentgen ray plate. Valuable time however should not be wasted in attempts to make a refined anatomic diagnosis. Diffuse rigidity denotes either intra abdominal hemorrhage or perforation and is a definite indication for immediate laparotomy.

Blood in the catheterized urine signifies kidney concussion or laceration. If no urine is obtained a few ounces of warm boric acid solution should be injected into the bladder. Failure of the fluid to return indicates bladder rupture.

Gun shot or stab wounds of the abdomen demand immediate exploration. The course of the bullet is generally determinable if a point of exit occurs and the surgeon may anticipate the probable lesions in the absence of exit the pathology is only conjectural. Bullet wounds through the epigastrium often cause perforation of the transverse colon stomach pancreas and diaphragm. In suspected damage of the pancreas careful examination of the viscus should be made by opening the gastrohepatic omentum or transverse mesocolon as overlooked pancreatic perforation is usually lethal. The small intestine should be examined throughout its entire length as multiple perforations are common. In low abdominal wounds the bladder sigmoid or rectum may be perforated. In stab wounds the lesion is generally focal and confined to the superficial viscera.

Hemorrhage may occur from the rupture of a single vessel multiple vessels or a solid viscus. In crushing injuries the bleeding areas are frequently multiple. Intestinal mesenteric and omental. The hemorrhage from a guillotined liver or spleen is commonly profuse.

**Prognosis.** The immediate outcome depends chiefly upon the degree of shock and hemorrhage. Extensive bleeding may cause almost immediate death. About one-half the cases of perforation recover if operated upon within six hours after twenty four hours few survive. In rare instances a small perforation may become spontaneously occluded and eventuate in recovery or abscess formation. Fecal fistulae developing from high lesions produce rapid emaciation whereas those of the large bowel usually close spontaneously. Pancreatic damage is especially serious and may cause considerable digestion of the abdominal wall during convalescence.

**Indications for Operation.**—In the presence of definite signs of intra abdominal hemorrhage or perforation or of a crescentic subdiaphragmatic air bubble immediate operation is imperative.

Involuntary muscle spasm with or without rebound tenderness is also a positive indication for laparotomy. Cases in which doubt exists after careful examination and observation are best explored; an overlooked perforation almost always eventuates in septic peritonitis.

**Preoperative Treatment** In the presence of grave shock surgical intervention is best withheld temporarily. The patient should be placed in bed with shock blocks at the foot and wrapped in warm blankets surrounded by hot water bottles. Transfusion is the ideal therapy for shock as a substitute. 10 per cent glucose in physiologic saline may be administered by phlebotomy. After the diagnosis has been determined morphine or its derivatives should be given hypodermatically to allay pain. In cases of profuse hemorrhage the lower extremities may be bandaged from below upward to conserve the depleted blood stream. The advisability of abdominal pressure is questionable; in the presence of perforation it is contraindicated.

**Operation** The exploration should be well planned and speedily but thoroughly performed; prolonged procedures are badly borne. If ether anesthesia is usually employed spinal is inadvisable. During the operation a continuous infusion of saline solution with 5 per cent glucose is supportive; a few minims of adrenalin hydrochloride may be added thereto. Unnecessary exposure to chilling should be carefully avoided.

The site of approach may be determined by focal signs; in their absence a mid split rectus incision is performed sufficiently large to permit of thorough exploration. Warm moist pads should be at hand to prevent evisceration. Bleeding vessels are promptly ligated. Damage to a large mesenteric trunk may require intestinal resection. Hemorrhage from a ruptured liver may be controlled by pressure with hot pads, searing with the cautery or by mattress sutures passed on round needles and tied lightly. Splenic injuries are ill suited to suture and splenectomy is generally preferable. Damage to the pancreas necessitates drainage.

Perforations of the intestines are closed by inversion, the suture line being reinforced with an omental tab. In cases of severe intestinal damage segmental resection is safer than extensive plastic repair. Following the closure of gut perforations most surgeons elect intraperitoneal drainage. The drains should be inserted so as not to contact the former site of leakage.

After completion of the peritoneal toilet the omentum should be reinspected for bleeding points as omental oozing exhibits little tendency to spontaneous hemostasis. In the absence of perforation resection of damaged portions of the omentum is unnecessary as they become rapidly revascularized.

Bladder perforations are closed by inversion suture and indwel

ing catheter is then passed through the urethra and retained for two weeks. Kidney lesions are subject to temporization. A totally crushed organ will require removal; less serious lesions may be treated conservatively as the kidney possesses considerable inherent reparative power. Occasionally a late hydroperinephrosis develops.

**Postoperative Treatment** Hemorrhage cases are replaced in bed in the shock position and those with perforation in the Fowler posture. Body warmth is maintained by blankets and hot water bottles and ample saline solution is administered through hypodermoclyses or infusions. Camphor and caffeine may be helpful stimulants during the first twenty-four hours. Blood transfusions are of greatest value.

## MESENTERIC LYMPH NODE DISEASE

### (MESENTERIC LYMPHADENITIS)

Although there are many possible explanations of the etiology and pathogenesis of lymph node disease, modern thought emphasizes the importance of considering the affection as a clinical entity. The adenitis may be secondary to focal intra-abdominal pathology or to infection elsewhere, especially of the upper respiratory tract and tuberculosis appears to be a causative factor in only a minority of cases.

**Anatomy** Meid has shown that the number of mesenteric nodes at birth may vary from 30 to 300. They are arranged in three groups as follows: the first are situated near the last anastomosing branches of the mesenteric vessels before the intestines are reached; the second in the vicinity of the next larger anastomosing branches; and the third at the root of the mesentery. Those at the mesenteric root are the largest and toward the intestines the glands tend to become progressively smaller.

**Lymph Node Infection**—The investigation of Arnold and others indicate that many organisms penetrate the intestinal mucosa and are destroyed by phagocytes in the mesenteric lymph nodes and liver. It also seems probable that the nodes may become invaded through hematogenous infection. Although substantiation of recent experiments showing the occurrence of blood stream infection with pathogenic organisms in otherwise normal individuals is not constant, the teachings of Adams, Alvarez and Heyd emphasize the consideration of subinfection from the gastro-intestinal tract as a cause of many syndromes, not the least of which is mesenteric lymph node involvement. Here as in many disease processes sensitization is probably a factor.

**Protective Mechanism**—The ability of the lymph nodes to filter out pathogenic organisms is well illustrated by the experiments of Drinker. He concludes a lymph node will filter practically all the

bacteria that enter it provided it is not massaged nor compressed. The empirical practice of advising absolute rest in the presence of acute superficial adenitis is in accordance with such views. Following this line of thought, the administration of cathartics in the suspected presence of mesenteric adenitis would be harmful through the production of hyperperistalsis and straining at stool. From the wealth of experimental evidence it appears that the mesenteric lymph nodes not only absorb bacteria, their toxins and other substances from the gastro-intestinal tract, but also those from certain generalized diseases and metabolic disorders.

**Etiologic Factors** —The mesenteric nodes are best considered as an integral part of the lymphatic system rather than a separate entity, and many agents other than focal infections may be associated with or cause their enlargement. Upper respiratory infections, influenza, acute anterior poliomyelitis, the acute exanthems, especially scarlet fever, typhoid fever and certain ulcers of the intestine, granulomatous diseases such as tuberculosis, syphilis, actinomycosis and Hodgkin's disease, malignant tumors and leukemia, deficiency diseases such as rickets, scurvy, pellagra, and others, as well as status lymphaticus and certain cases of intestinal stasis. Many of these exhibit their greatest effects through the gastro-intestinal tract. *Tuberculosis, formerly considered the sole etiology, plays but a minor rôle.*

**Age Incidence** —Foster has made a study of 123 case records of mesenteric lymph node disease from the files of the New York Post-Graduate Hospital and Medical School of Columbia University from 1914 to 1936, during which time appendicitis was diagnosed in the same institution in 16,964 cases. There were 66 patients under sixteen years of age and the highest incidence of the disease occurred between the ages of seven and ten years. Apparently the pathology is rare during the first year of life.

**Association With Chronic Appendicitis** —A startling finding is the close relationship between mesenteric adenitis and chronic appendicitis, and also the fact that fecoliths occurred in 47 per cent of the appendices which were associated with adenitis. In most instances the adenopathy was evidenced chiefly in the iliocecal group of nodes. It is also worthy of note that mesenteric node disease occurred as the sole pathology in only 3 cases and that none exhibited malignancy.

In the entire group some evidence of appendiceal disease was grossly present in 95 per cent, and in 88 per cent this was confirmed by microscopic examination. Upper respiratory or focal mouth infections occurred in 37 per cent. In the vast majority the adenitis was chronic in type, being purulent in only 3 cases. Although 32 per cent were recorded as tuberculous, this diagnosis in most instances was clinical.

**Symptomatology**—Recurrent abdominal symptoms were a striking feature in 80 per cent of the cases. Vomiting occurred in 50 per cent, tenderness in 69 per cent, muscle spasm in 56 per cent, rigidity in 16 per cent, an abdominal mass in 10 per cent, and distention but rarely. The temperature varied from  $100.2^{\circ}$  to  $101^{\circ}$  F. and the average white blood cell count was 11,800.

**Diagnosis**—Acute mesenteric lymph adenitis cannot be accurately differentiated from acute appendicitis and the subacute and chronic types may mimic closely the right lower quadrant syndrome of chronic appendicitis. Mesenteric nodal pathology should always be considered, however, when one or more of the following are present: (1) Recurrent attacks of abdominal pain; (2) familial history of tuberculosis or the finding of tubercles in the patient; (3) the history or findings of upper respiratory or focal mouth infection, constipation, visceroptosis, pinworms, round worms or amebic dysentery; (4) enlargement of the surface lymph nodes from whatever cause; (5) positive roentgenologic findings of calcified mesenteric lymph nodes or appendiceal stasis; and especially (6) any case lacking the typical sequential symptomatology of appendicitis.

**Tuberculin Test**—The majority of individuals exhibit a positive tuberculin test after the age of five years. A positive finding in early life does not indicate an active tuberculous infection unless the infection is the first one or the test is done a certain time after the onset of the first infection. A negative test repeated in a day or two and still found negative during an acute illness such as acute adenitis may indicate that the infection is tuberculous if another test a few weeks later is positive. After the fifth year a positive reaction is comparatively worthless.

**Treatment**—Except in the presence of lymph node excision nothing beyond appendectomy is required. The promiscuous removal of a lymph node for biopsy is a dangerous procedure. Acutely inflamed nodes may harbor virulent streptococci and the excision of a chronic node may result in circulatory damage to the intestine.

Following the postoperative convalescence a hygienic dietetic regimen should be inaugurated, supplemented by heliotherapy and the administration of viosterol or fish liver oils. All foci of infection should be removed and faulty health habits corrected. The abolition of bacterial sensitivity may be indicated in specific instances.

## CHAPTER XXVIII

### GASTRIC ANOMALIES

ALTHOUGH stenosis of the pylorus due to hypertrophy of the sphincter muscle occurs quite frequently, complete occlusion or atresia is exceedingly rare. Hour-glass stomach, transverse septum across the organ and total gastric atresia are pathologic curiosities. Duodenal occlusion may be associated with the gastric anomaly.

#### CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

There are two definite factors in the pathogenesis of congenital pyloric stenosis: (1) *Pylorospasm*, dependent upon tonic spasm of the pyloric musculature, and (2) *stenosis*, resulting from congenital hypertrophy of all the tissues, but especially of the circular muscular fibers. The two types of cases produced thereby are not sharply distinguishable. Most observers consider hypertrophy to be the primary causative factor and spasm a resultant secondary reflex. The reverse is untenable for spasm of an involuntary muscle is incapable of producing hypertrophy. Swelling of the mucous membrane may be an added factor.

In most instances the sequence of symptoms indicates that for a time the propulsive powers of the stomach are sufficient to overcome the primary hypertrophic stenosis, and that finally a large element of spasm develops and closes the pylorus. This hypothesis is substantiated by cases in which the tumor persists without symptoms after the spasm has been relieved. In others, however, spasm is the dominant or sole element, as is evidenced by numerous medical recoveries, and by the fact that in exceptional cases no tumor or true stenosis is found at operation or necropsy. It should be emphasized that in the vast majority of cases operated upon, definite organic hypertrophy is readily demonstrable.

**Incidence**—The disease occurs most often in boys (approximately 80 per cent) and in more than one-half of the cases the first born is affected. At times there is a familial element and more than one child is afflicted, it has also occurred simultaneously in twins. Negroes are relatively immune.

**Etiology**—The cause is undetermined. Some investigators attribute it to a hypertonic imbalance of the vegetative nervous system, and others to various factors: fetal allergic sensitization,



**Symptomatology**—Developing in an apparently normal healthy infant, the disease generally manifests itself between the first and fifth week after birth. Evidence, however, may be manifested in the first few days of life (4 per cent), and in rare instances the affection develops after the second year, or later. In all cases there is probably a combination of spasm and stenosis.

**Vomiting** is the dominant symptom. At first uncharacteristic and only occasional, it steadily becomes more frequent and of projectile type. Emesis may occur after each feeding or some hours later, and with dilatation of the stomach more food may be vomited than is ingested. The vomitus consists of undigested milk and often

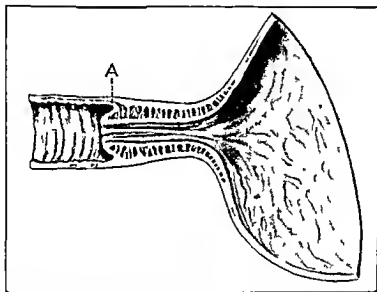


FIG 156—Congenital hypertrophic pyloric stenosis. Note the abrupt ending of the hypertrophy at the duodenal junction. A indicates the site at which accidental perforation may occur during operation.

contains mucus and occasionally blood streaks. Hyperchlorhydria may or may not be present.

Constipation is marked, its degree depending upon the pyloric patency.

Loss of weight is striking in severe cases but in mild conditions the weight may be surprisingly sustained.

Visible peristalsis is a characteristic symptom which is seldom absent. The gastric waves are exhibited especially after feeding and pass from left to right, usually in groups of two. They apparently cause no pain and may occur during sleep. In many instances a movable pyloric tumor, the size of a small olive, is palpable slightly to the right of the mid line just under the liver edge, or lower

The epigastrium may be distended in contrast to the scaphoid abdomen

Gastric retention may be demonstrated by passing a stomach tube or by a barium roentgenogram. A four to eight hour residue occurs frequently and several hours may elapse before any barium enters the intestine

Dehydration is marked in severe cases—sunken eyes, pinched expression, dried tongue and putty like inelastic skin. (Refer to Dehydration). Alkalosis may result from the loss of chlorides in the vomitus. The urine is scant, high colored and concentrated. Fever may occur in the late stages.

**Diagnosis**—The chief diagnostic factors are the early development of symptoms (before six weeks), obstinate projectile vomiting, visible gastric peristalsis and palpable tumor. The last is inconstant. A barium roentgenogram is seldom needed for diagnosis. In pyloric or duodenal atresia the symptoms develop immediately after birth and in the latter condition the vomitus may contain bile. In esophageal occlusion the food is regurgitated at once in an unchanged condition and visible peristalsis is wanting.

The distinction between cases of pylorospasm and those with dominant organic hypertrophy is made chiefly by the earlier onset of symptoms in the latter, the more pronounced obstruction and especially by the lack of prompt and adequate response to medical treatment.

**Prognosis**—Recovery without operation is more probable when the symptoms develop late, in such cases the element of spasm is apt to be dominant and that of stenosis minimum. Whereas the mortality of the older types of operations such as gastro-enterostomy and complicated pyloroplasty was over 50 per cent, the Fredet-Rammstedt pyloromyotomy has reduced the average to approximately 15 per cent. Most deaths occur in starved emaciated infants under 3 pounds; in favorable early cases the surgical mortality is less than 5 per cent.

**Medical Treatment**—Medical treatment by a competent pediatrician should always be given a trial. This comprises (1) efforts to stop vomiting, (2) proper feedings, and (3) the prevention or treatment of dehydration, alkalosis and hypochloremia. A daily lavage with 1 per cent saline solution is sometimes helpful. Atropine is specifically indicated and should be administered in large doses. Beginning with  $\frac{1}{1000}$  grain every four hours, the dosage should be increased rapidly until skin flushing and dilatation of the pupils result. Tolerance is soon established and large doses are well borne. If vomiting occurs too frequently, the medication should be given hypodermically. Papaverine hydrochloride is also recommended.

Breast feedings should be continued and promptly repeated if vomited. Supplementary feedings of 10 per cent rice flour or arrow

root may prove beneficial. In prevent dehydration and hypochloremia small amounts of saline solution should be given rectally supplemented by hypodermoclyses of 3 per cent glucose in physiologic salt solution. (Refer to Dehydration.) The total fluid intake in twenty four hours should exceed 250 cc. Needless to say the amount lost through vomiting requires replacement.

Roentgen ray exposure of the thymus is futile. Frequent weighing is an excellent prognostic guide. *Unless definite improvement obtains within a week surgical intervention is urgently indicated before the patient becomes too weakened.* It should be emphasized that in a well established case there is little hope of cure by medical treatment.

**Surgical Treatment**—The simple and safe operation of pyloromyotomy (Fredet Rammstedt 1912) has superseded those of gastro-enterostomy and pyloroplasty. The immediate results in early cases are so satisfactory and the after progress so remarkably good that surgical therapy should be advocated without hesitancy. The operative mortality is chiefly the result of procrastination—a desiccated starved infant of  $4\frac{1}{2}$  to 5 pounds is an extremely grave risk.

**Preoperative Preparation**—Meticulous preoperative care will salvage many otherwise hopeless cases. Dehydration with accompanying alkalosis is best combated by repeated hypodermoclyses or phleboclyses of 3 per cent glucose in physiologic saline solution. (See Dehydration.) Preoperative blood transfusion is also of great value.

**Fredet Rammstedt Pyloroplasty**—Although the operation may be performed under 0.5 per cent novocaine-adrenalin anesthesia most surgeons elect light ether narcosis. The anesthetic is well borne and the operation may be done speedily without the danger of straining and evisceration. The child's chest and extremities should be well blanketed and kept warm during the procedure and a pad under the back is helpful.

The abdominal skin is sterilized with half strength tincture of iodine sponged with 95 per cent alcohol and dried. A 2 inch upper right pararectus incision made relatively high so as to be over the liver is deepened to the posterior rectus sheath. The latter with the peritoneum is meticulously opened between clamps during inspiration. The index finger is then hooked beneath the liver and the pyloric tumor withdrawn. Gripping the tumor between the left thumb and index finger an incision is made on its anterior wall in the long axis of the gut midway between the greater and lesser curvatures. Although the incision may be extended proximally beyond the pyloric canal it should end distally where the duodenum begins. (Fig 157.) Great care is required at this point lest the thin duodenal wall be perforated as the hypertrophied musculature ends abruptly at the pyloroduodenal junction.

*The incision should divide only the serosa and superficial muscle fibers the deeper fibers are then separated by blunt dissection until the mucosa bulges freely into the incision throughout its entire length (Figs 157 to 160) The operation is then complete Oozing points may be controlled by hot pads or ligation An ind-*

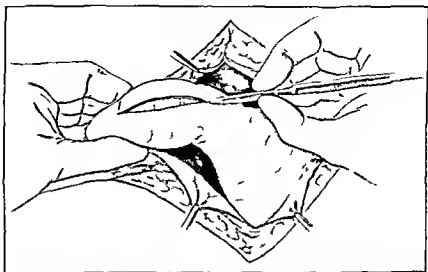


FIG 157 —Fredet Rammstedt operation The incision through the serosa begins at the pyloric vein and is carried proximally the entire length of the tumefaction

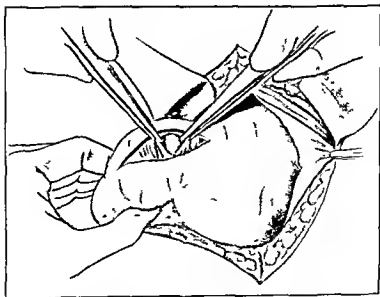


FIG 158 —The exposed circular fibers are separated by artery forceps

vertical perforation should be closed with No. 0 catgut or fine silk and be reinforced by sewing the omentum over it.

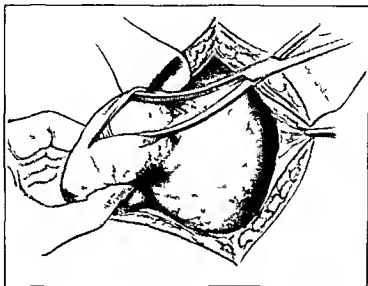


FIG. 159 The mucosa is exposed the full length of the pylorus

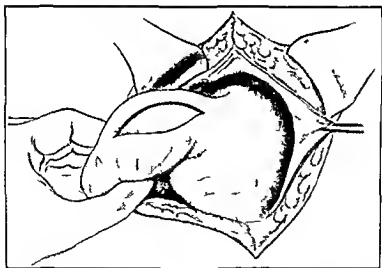


FIG. 160—Operation completed. A ligature has been applied to a branch of the pyloric vein.

The pylorus is dropped back into the abdomen and disappears under the liver. The peritoneum and posterior rectus sheath are accurately approximated with No. 1 plain catgut, the muscle and

anterior sheath with No 1 chromic and the skin with dermal suture or clips. Two retention sutures anchored by Davey buttons are advisable for added support. The gauze dressing is firmly secured with adhesive plaster and a tight abdominal binder is superimposed. Adequate abdominal support is essential for two weeks as young infants are especially subject to dehiscence.

**Postoperative Care**—After-care is extremely important and the cooperation of the pediatrician is invaluable. The child should be kept well blanketed and warm. An infusion of 150 cc. of physiologic saline solution containing 5 per cent glucose is an excellent preventative of postoperative shock. As soon as the patient recovers from the anesthetic 0.5 dram doses of warm water may be given by mouth every hour. On the second day breast feedings may be resumed if the child be too weak to nurse the mother's milk should be pumped and given with a medicine dropper. Inadequate fluid intake during the first few days should be supplemented by hypodermoclyses of saline solution with 3 per cent glucose. Proctoclyses are messy and unsatisfactory.

The postoperative convalescence is generally uneventful and by the second week full regular feedings are established. Sutures are not removed before the twelfth day. The symptomatic postoperative results are permanent. The children have excellent digestion and in all respect develop normally.

### PEPTIC ULCER

Peptic ulcer is very uncommon in children and the subject has received scant recognition. Morse found 5 cases of peptic ulcer in an analysis of 14,000 cases of chronic or recurrent abdominal pain. Palmer collected 45 cases which were demonstrated at operation. A correct preoperative diagnosis had been made in only 10. Wells reported a case of probable prenatal perforation and Butka a perforation occurring on the sixth day. Apparently more cases are discovered by the pathologist than the pediatrician.

**Symptomatology**—Most cases of primary peptic ulcer occur in prepubescent girls and the symptomatology generally simulates that of the adult type. In younger children however the signs and symptoms tend to be atypical and ulcer is not suspected. Epigastric distress and especially nocturnal pain which awakens the child and which is relieved by taking milk or crackers should arouse the suspicion of peptic ulcer. The pathology is readily demonstrable by roentgen ray examination. Hemorrhage and perforation occur but rarely.

**Syndrome of Perforation**—The symptomatology of acute perforation mimics that occurring in adults. Sudden agonizing pain generally accompanied by mild ephemeral shock, nausea and vomiting

and the immediate development of diffuse abdominal rigidity. In young children hyperesthesia of the skin may be more pronounced than rigidity. The child lies still afraid to move, the abdomen is carefully splinted and the respirations are shallow and of thoracic type. Rectal tenderness is present early. During the first few hours following perforation, the temperature may be subnormal, normal or slightly elevated, and the pulse only moderately accelerated, the leukocytosis averages 12,000 to 15,000 with slight polynucleosis. During the second twelve hours this stage of irritative chemical peritonitis progresses to that of diffuse suppurative peritonitis.

**Treatment of Peptic Ulcer**—Most peptic ulcers in children respond to a medical regimen of rest, diet and alkalis. Cicatricial stenosis with progressive duodenal obstruction rarely develops or requires surgical relief by gastro-enterostomy or pyloroplasty. Perforation demands immediate laparotomy; the perforation should be closed by purse-string inversion with No. 1 chromic catgut and the site of closure reinforced by an omental tab. A concomitant gastro-enterostomy should not be performed unless definite obstruction results from closure of the perforation. Most apparent stenoses disappear.

## INTESTINAL HEMORRHAGE

### (MELENA)

There are numerous conditions which may produce intestinal hemorrhage. The bleeding may be so slight as to be only demonstrable by chemical stool examination, more often the quantity is moderate in amount and in rare instances it may be profuse. Fresh blood from near the anus and rectum is bright red and unmixed with the stool, that from the small intestine, stomach, esophagus or pharynx is intimately mixed with the feces and imparts a tarry color (melena).

**Melena Neonatorum**—This condition occurs about once in every 1000 to 2000 births. The blood may come from the stomach and intestines (melena vera) or enter the stomach by swallowing the primary source being the nasopharynx or lungs (melena spuria). Lesions of the gastro-intestinal membrane or deeper vessels are the commonest cause but at times no definite pathology is demonstrable. The etiology is not clearly understood. The melena generally appears on the second to fourth day and the loss of blood is often sufficient to produce pallor and collapse. Although the hemorrhages seldom last more than two or three days they may prove fatal. Specific treatment comprises the transfusion or intramuscular injection of whole blood, horse or rabbit serum is less effective. (Refer to Transfusion.)

**Pathology** The pathology of Meckel's diverticulum depends in part upon certain anatomic and embryologic factors. An important consideration is the size of the opening of the diverticulum into the ileum. A diverticulum with a large ileac ostium readily permits of the entrance and exit of the fecal stream. Although in unusual instances foreign bodies may lodge therein, this type of the anomaly generally remains asymptomatic. Conversely, a diverticulum exhibiting a narrow opening into the ileum is subject to fecal stasis

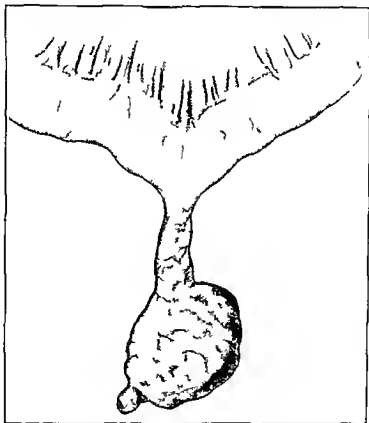


FIG. 161. Gangrenous Meckel's diverticulum.

impaction, inflammation, gangrene, perforation, or intestinal obstruction (Fig. 161). At times the pouch may become gradually distended and reach such proportions as to produce torsion of the bowel. Infrequently a diverticulum may form part of the contents of a hernia.

**Diverticulitis**—The diverticulum may be the site of a purely inflammatory process. The pathology differs in no way from that of acute appendicitis, and the process may be catarrhal, suppurative, gangrenous, or perforative. Although the sequential symptomatic



ogy may mimic that of appendicitis the focalization of pain, tenderness and rigidity tends to be nearer the umbilicus. Phlegmonous diverticulitis may be complicated by pyelophlebitis and result in secondary hepatic abscess.

**Ulcerative Diverticulitis**—The dystrophic gastric mucosa cells secrete hydrochloric acid and at times produce ulceration which genetically resembles peptic ulcer. The ulceration leads to pain and in some instances to bleeding with resulting melena and secondary anemia. Perforation with localized or diffuse peritonitis may follow.

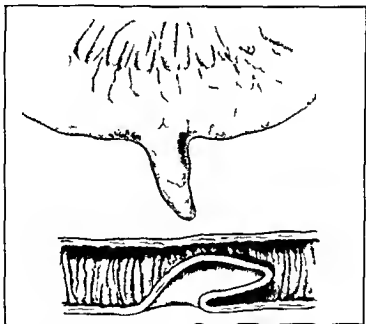


FIG. 169. Intussusception of a Meckel diverticulum.

**Patulous Diverticulum**—Very rarely a patulous tubular process opens at the umbilicus. Depending upon whether its lumen is patent or partially occluded the discharge will be fecal or mucous. In other instances a granulomatous formation at the umbilicus may be the only indication of an incompletely obliterated omphalo-mesenteric duct. Secondary closure of the umbilical ostium may result in cyst or abscess formation. Cases have also been reported in which the intestine projected through the diverticulum.

**Intestinal Obstruction.**—Constriction of the intestine by either the diverticulum or its residuum is one of the most common lesions associated with Meckel's diverticulum. At times the diverticulum remains connected to the umbilicus through the persistence of a

fibrous cord or the latter may attach it to some other organ. This potentially dangerous mechanism may cause intestinal obstruction through angulation, constriction or torsion of a bowel segment.

**Volvulus**—Torsion of the diverticulum itself is exceedingly rare. More often volvulus of the ileum occurs about the diverticulum acting as a fixed point of rotation.

**Invagination of the Diverticulum**—A short diverticulum may become invaginated within the lumen of the intestine (Fig. 162) and this may be followed by an intussusception of the ileum (ileo-ileic). The condition is rare and seldom develops before late childhood whereas ordinary intussusception occurs in early infancy. The obstruction is usually incomplete and produces little alteration in the vascular competency of the bowel. Bloody mucus is recordingly trifling or absent and the clinical syndrome is that of intestinal obstruction.

Meckel's diverticulum may rarely be the site of tuberculous or typhoid ulceration which may eventuate in perforation. The former is generally associated with intestinal tuberculosis and the latter occurs from involvement of the Peyer patches in the diverticulum.

**Neoplastic Growths**—These are of very rare occurrence and may be either benign or malignant. The reported cases include adenoma, sarcoma, carcinoid, disease, medullary carcinoma and myxoma.

**Symptomatology**—From the foregoing discussion of the pathology it is evident that the symptomatology may be extremely variable. The vast majority of diverticuli remain asymptomatic throughout life.

Acute inflammatory processes mimic closely the syndrome of appendicitis and the condition is generally so diagnosed. The focalization of signs near and to the right of the umbilicus should arouse suspicion of Meckel's diverticulum. In cases of intestinal obstruction of undetermined origin Meckel's diverticulum should always be considered as a possible factor. Halsted estimates that the anomaly accounts for approximately 6 per cent of all cases of intestinal obstruction.

**Melena** at times may be the only sign. The blood is usually dark in color but may be bright red and profuse. The pain accompanying the ulceration of heterotopic tissue is strikingly similar to that of gastroduodenal ulcer. It is cyclic in character and exhibits not only a periodicity in relation to the ingestion of food but also a hunger type of distress. This is accounted for by the fact that the secretion in the diverticular mucosa parallels that in the duodenum.

**Diagnosis**—Although a definite preoperative diagnosis is often impossible Meckel's diverticulum may at times be diagnosed with reasonable certainty. Melena or intestinal hemorrhage should always arouse the suspicion of a diverticulum. Other conditions such as hemophilia, purpura hemorrhagica, hemorrhagic disease of

of the new born and certain conditions associated with splenomegaly must be excluded. In the acute inflammatory type which closely resembles appendicitis Meckel's diverticulum should be sought for when the paracecal pathology is inadequate to explain the symptoms.

Roentgenologic evidence may be of definite diagnostic value. At times the diverticulum may reveal itself in a routine gastro-intestinal series. When the condition is suspected however the roentgenologist should be informed so that he may devote his attention specifically to small intestine study. (A different technic and contrast mixture is usually employed.)

**Prognosis** The outcome following surgery is influenced greatly by the severity of the pathology. Wellington reports a mortality of 40 per cent in a collected series of 396 cases. The high death rate is attributable to several factors: the liability of diverticulitis to early perforation with slight tendency to focalization of the infective process; frequent involvement of the bowel; and the mechanical difficulties encountered in removing the diverticulum. In Goodman's series of 23 cases from the surgical services of the New York Post Graduate Hospital (1930-1936 inclusive) there were 2 deaths from bronchopneumonia and postoperative ileus respectively (8.7 per cent.)

**Treatment**—Diverticulectomy is indicated in all cases of Meckel's diverticulum which produce symptoms. When however the anomaly is asymptomatic and is accidentally discovered during laparotomy the advisability of routine removal is questionable. When one considers the common incidence of Meckel's diverticulum it is at once apparent that only a very small proportion of the cases ever produce symptoms. Moreover diverticulectomy is associated with a relatively high mortality. It would therefore seem best not to remove the diverticulum unless its anatomic pattern indicates a potential hazard. Wide-mouthed pouches which empty freely and exhibit no evidence of bands or peridiverticulitis should be left undisturbed. Such types seldom produce symptoms and their removal leaves a large ostium whose closure may damage the luminal capacity of the gut. Conversely diverticuli with narrow necks are definitely liable to inflammation. Such types should be removed; moreover the plastic closure of the small ostium is a simple and safe procedure.

**Operative Technic**—Following amputation of the pouch the treatment of the opening into the intestine will depend upon its size. Small ostia may be closed by simple inversion and suture of the bowel edges. Large openings are best treated by approximating their edges in the longitudinal axis of the gut so as to obviate luminal stenosis. The suture line of No. 1 chromic catgut should be reinforced by a continuous Lembert of No. 0 plain catgut. The lumen

of the gut should always be tested following the plastic repair, in rare instances a lateral anastomosis may be required for adequate patency. Abscess crises require drainage.

### ENTERIC CYSTS

Cysts developing in the wall of the intestine are very unusual. They project into the lumen and may cause intestinal obstruction directly or form the apex of an intussusception. The cysts may be single or multiple, occur most often in the ileocecal region and seldom attain large size.

**Treatment**—Although in some instances the cysts are removable through an enterotomy, most cases require resection of the involved gut.

### FOREIGN BODIES IN THE ALIMENTARY TRACT

Infants have a tendency to put small objects into their mouths and occasionally swallow them, coins, buttons and pins being common offenders. Smooth objects pass readily through the esophagus into the stomach and produce no symptoms except coughing or discomfort during the act of deglutition. Pointed objects such as pins or open safety pins also pass freely when the dull end is directed distally.

**Esophageal Impaction**—The common location for the lodgment of a large foreign body in the esophagus is either at the beginning of the organ or where it is crossed by the left bronchus. The site of impaction may be determined by passing an esophagoscope or catheter or by a roentgenogram. Unless regurgitated the object should be removed through the esophagoscope. Objects which have traversed the esophagus into the stomach even when as large as a twenty five cent piece usually pass on and ultimately become voided in the feces.

**Foreign Bodies in the Stomach**—These may be vomited. When retained they seldom produce any symptoms whatsoever. Smooth substances generally pass into the duodenum with the next meal although at times they may be arrested for several days. It is quite remarkable that objects which are apparently larger than the pylorus are nevertheless extruded into the duodenum. Amylaceous foods such as bread, potatoes and cooked cereals may aid in their coating and propulsion. Sharp pointed objects occasionally impinge in the gastric mucosa and produce irritation, ulceration and rarely perforation. Serial fluoroscopic or roentgen ray examinations should be made in such cases if the object remains stationary; it is probably impacted and will require mechanical removal either through the esophagoscope aided by the fluoroscope or by gastrotomy.

Hair balls occasionally accumulate in the stomach from the swallowing of hair, fur or wool from blankets. They may attain large size and form a palpable tumor.

**Foreign Bodies in the Intestine** — Once a foreign body has entered the intestine, its further progress is usually uneventful. Sharp objects, however, may become arrested at the ileocecal valve, permanent lodgment necessitates operative removal. Fish bones, seeds and splinters may impinge in the crypts of Morgagni and produce pain and tenesmus. Unless promptly removed, infection and abscess formation may result.

**Prognosis** — Most foreign bodies are evacuated within a few days. For their detection the stools should be liquefied with water and strained through cheesecloth. In the case of smooth bodies such as coins or buttons, the parent's anxiety far exceeds the gravity of the condition.

## CHAPTER XXIX

### INTESTINAL OBSTRUCTION

#### INTUSSUSCEPTION

INTUSSUSCEPTION is predominantly a disease of infancy and is the chief cause of intestinal obstruction in children under five years of age. Approximately one-half the cases occur during the first year of life and most of the remainder during the second year. The condition is twice as frequent in males.

**Etiology**—Most cases develop in healthy infants. Hyperperistalsis in association with unusual mobility of the cecum appears to be a contributory factor. There may also be an added element of disturbed innervation which incites irregular overactive peristalsis. Furthermore the ileum and colon are nearly the same diameter in early life and such anatomic relationship may predispose to incompetency of the ileocecal valve. Other accredited predisposing causes comprise diarrhea constipation colic Meckel's diverticulum appendiceal irritation invagination of the appendix intestinal ulceration foreign bodies including fecal masses and parasites polypi and especially swelling of the lymphoid tissue about the ileocecal valve.

**Pathology**—One portion of the gut invaginates into another and the process is almost always direct the proximal segment or *intussusceptum* slips into the distal or *intussusciens* dragging the mesentery with it. A retrograde type occurs very rarely. Once started the intussusception is continued by the passage of the original invagination along the lumen of the bowel. The distance which the intussusception may invaginate into the intussusciens is limited by the mobility of the invaginating gut in rare instances the apex may protrude from the anus. The drag upon the mesentery causes the mass to assume a curved sausage-shaped form with the concavity directed toward the umbilicus.

Although the bowel may remain patulous and its circulation be maintained this seldom occurs. Pressure upon the mesentery produces venous and lymphatic stasis with resulting edema. Circulatory damage usually follows and the lumen of the acutely inflamed intussusceptum becomes completely obstructed. Such severe congestion produces bleeding into the bowel infarction may also develop and cause further hemorrhage. Irreducibility commonly results from adhesive inflammation between the adjacent serous

surfaces. With continued circulatory damage perforation may occur or the entire process may become gangrenous.

Spontaneous reduction occasionally occurs in the early stages of intussusception. In unusual instances the gut remains viable and patent and the intussusception pursues a chronic course. Very rarely sloughing of the mass and its extrusion per rectum has resulted in cure.

**Varieties** 1 *Enteric or Ilac*—This type is usually agonic or postmortem. The intussusception is often multiple or compound and the jejunum is frequently involved. As a clinical entity it is very rare. (Fig 163.)

2 *Colic*—The colon alone is rarely involved in early life. (Fig 164.)

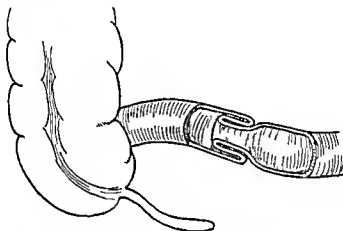


FIG 163 Enteric variety of intussusception in which the ileum invaginates into itself (ileoileal)

3 *Ileocolic*—The ileum is invaginated through the ileocecal valve in about 90 per cent of the cases. The cecum may be secondarily involved but the ileum persists as the apex of the intussusceptum. (Fig 165.)

4 *Ileocecal*—This type occurs in approximately 80 per cent of the cases, being especially predominant in young infants. The cecum with the ileum behind it evaginates into the colon and the ileocecal valve occupies the apical position. (Fig 166.)

Mixed types occasionally occur especially double intussusception. Retrograde or ascending intussusception and chronic forms are rare in children.

**Symptomatology**—The cardinal symptoms comprise (1) Recurrent paroxysmal attacks of extreme abdominal colic alternating with periods of freedom from pain. (2) tenesmus with the passage of blood stained mucus. (3) the development of an abdominal tumor.

and (4) progressive **obstipation**. A previously healthy infant is suddenly seized with severe abdominal cramps, draws up its knees, becomes pale, and either holds its breath in an attempt to move the bowels, or shrieks with pain. Vomiting generally occurs.

The initial seizure is ephemeral in character and in a few minutes the acute pain subsides and the child is comparatively comfortable. A normal bowel movement may occur with the onset. After a vari-

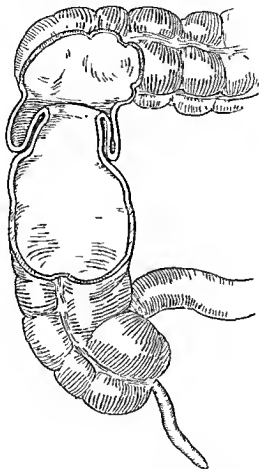


FIG. 164 —Colic variety of intussusception. The colon invaginates into itself.

able period of time from a few minutes to an hour or more, the paroxysmal pain recurs. Each attack is accompanied by temporary collapse and tenesmus. *Recurrent rectal tenesmus with an empty bowel is very suggestive of intussusception.*

In a few hours, and generally within twelve, bloody mucus is passed. The invaginated bowel may be felt, and sometimes seen, as an elongated tumor along the course of the colon, at first on the



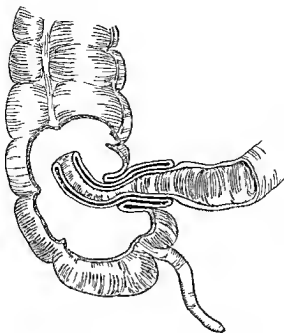


FIG 165 —Ileocolic variety of intussusception. The invagination begins in the terminal ileum and the intussusception passes through the ileocecal valve into the cecum and ascending colon at the expense of the small gut.

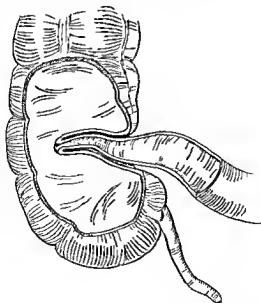


FIG 166 —Ileocecal variety of intussusception. The ileocecal valve forms the head of the intussusception and the invagination is at the expense of the large bowel.

right side, then in the epigastric region, and finally on the left. After twelve hours the tumor may be palpable upon rectal examination, in advanced cases the apex of the intussusception feels like the cervix uteri, and in extreme cases it may present at the anus.

**Pain**—The pain is most intense in the early stages and the paroxysms are frequently accompanied by temporary pallor and at times collapse. In the intervals between paroxysms, the child is apt to be uncomfortable. In some instances, however, there is complete freedom from discomfort. Some years ago at the New York Post-Graduate Hospital, the writer demonstrated such a case to a group of matriculates. While the operating room was being prepared the infant lay in its crib, happily playing with a rattle. There was a fourteen-hour history of recurrent attacks of colic during which the child paled and strained to move its bowels. The appearance of bloody mucus alarmed the mother who brought the child to the hospital. A tumor was readily palpable in the right upper abdomen, extending across and above the umbilicus, yet to all appearances the child seemed perfectly well. At operation a 5-inch ileocecal intussusception was reduced. Recovery was uneventful.

**Tumor**—A tumefaction is demonstrable in the majority of cases within twelve hours. Distention, occurring the second or third day, may later mask it. Palpation reveals the tumor on the left side in over one-half of the cases; in about one-third it may be felt rectally. The mass may alternately harden and soften with peristalsis, and if a paroxysm occurs during rectal examination, it may be felt to enlarge and approach the anus. A rapidly advancing intussusception may reach the anus within forty-eight hours, when protruding it has the appearance of a rectal prolapse.

**Stools**—The earlier paroxysms are often accompanied by one or more normal stools, later by bloody mucus. Complete obstruction may occur within a few hours but usually not until the second or third day. Distention, visible peristalsis and stercoraceous vomiting are late symptoms. The temperature remains normal or subnormal, and the pulse increases as absorption and circulatory damage progress. Leukocytosis and polynucleosis are moderate.

The syndrome of recurrent paroxysms of crampy pain, accompanied by rectal tenesmus and bloody mucus, occurring in a previously healthy child, is highly presumptive evidence of intussusception. In the absence of a palpable tumor, a flat roentgen-ray plate, made without any preparation, will generally exhibit the distended intestinal coils above the obstruction.

**Atypical Cases**—In atypical cases, the symptoms may be so mild that the condition will be overlooked if attention is not directed to the character of the bowel movements and to the presence of a tumor. In older children the disease may be of a more chronic

nature gradual onset less severe pain and vomiting and incomplete obstipation When the mesenteric circulation is undamaged bloody mucus does not occur and there may be diarrhea The symptoms often suggest catarrhal colitis until strangulation suddenly develops

**Clinical Course** In exceptional cases the invagination reduces spontaneously and all symptoms disappear Recurrence is not unusual The vast majority of cases however progress to a fatal issue Death results from shock intestinal obstruction or peritonitis There are a few recorded recoveries in which the gangrenous intussusception was extruded per rectum

The average duration of neglected fatal cases varies from one to several days Chronic cases may live for months and die from exhaustion Intussusception occasionally recurs after operative reduction and the catastrophe generally develops within forty-eight hours

**Diagnosis**—The cardinal diagnostic symptoms of sudden onset recurrent proximal pain vomiting and tenesmus with bloody stools develop in the absence of fever The blood streaked mucus may be erroneously attributed to *colitis* Conversely colitis may be mistaken for intussusception In the former diarrhea occurs early persists and is accompanied by fever in the latter a tumefaction is pathognomonic *Meckel's diverticulitis* is occasionally accompanied by bloody stools the inflammatory mass which develops near the umbilicus is associated with fever and tenesmus is absent In very rare instances of appendicitis there may be blood streaked stools however the sequential symptomatology and physical signs are focalized in the right lower quadrant *Henoch's purpura* may suggest intussusception evidences of purpura are exhibited elsewhere in the skin and joints An intussusception presenting through the anus may be mistaken for *rectal prolapse* The antecedent acute history and presence of an irreducible mass is diagnostic

**Prognosis**—Practically all neglected cases succumb Operative statistics are chiefly influenced by the integrity of the gut Cases operated upon within a few hours of onset have a minimum and but slight mortality between twelve and twenty four hours approximately 70 per cent recover and between twenty four and forty eight hours 50 to 60 per cent After the second day the mortality increases rapidly gangrenous cases requiring resection rarely survive likewise those with perforation

**Treatment**—Invaginations rarely reduce spontaneously and palliative measures comprising the administration of belladonna and colonic pressure injection are seldom effective The vast majority of cases require immediate surgery

**Colonic Injection**—While the operating room is being set up, colonic injection may be attempted following the preliminary administration of atropine. The child should be anesthetized. While the hips are elevated warm water or olive oil is slowly injected into the rectum by gravity, the syringe being held not more than 5 feet above the buttocks. If successful the tumor will disappear and gas and liquid feces will be freely expressed. Prolonged temporization is interdicted. The procedure usually fails and immediate laparotomy should follow. If there is much prostration preoperative blood transfusion or the infusion of 5 per cent glucose in physiologic saline is of great benefit.

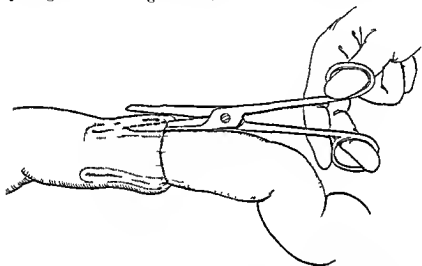


FIG. 107—Division of the neck of the intussusception in cases of irreducibility

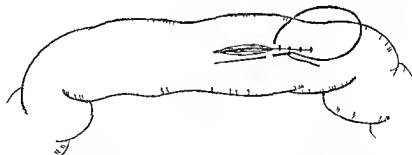


FIG. 108—Closure of the ileotomy following reduction of the intussusception

**Operation**—The abdomen is opened through a mid right rectus incision and the intussusception is delivered into the wound. Reduction should be attempted by pushing the invaginated portion out of the sheath instead of pulling upon it. Edema at the neck of the sheath and of the invaginated gut, rather than adhesions is

arterial supply soon follow with resulting gangrene and perforation. Thrombosis of the mesenteric vessels may accompany the process.

**Symptomatology** — Acute volvulus is the usual type. The onset is sudden with severe abdominal pain, nausea, vomiting and the prompt development of a mass. Accumulation of gas in the torsioned loop is constant and rapid, producing a tender, tense, tympanitic tumefaction. The pain is very severe, of paroxysmal colicky type, with remission periods of partial relief. At the onset there may be a copious bowel movement; subsequent obstipation is complete, however, and enemas are returned without feces or flatus. Tenesmus and bloody mucus may occur, although less frequently than with intussusception. The pulse is rapid at the outset but slows somewhat after the initial shock subsides. The latter varies in ratio to the magnitude of torsion and circulatory disturbance.

Depending upon the viability of the gut, the symptoms are those of mechanical intestinal obstruction, with or without strangulation. Auscultatory peristalsis is increased above the rapidly produced tympanitic mass, and in subacute cases peristaltic waves may be seen. The location of the tumefaction depends upon the site of the pathology, and during a period of several hours the mass may change in position. In the late stages general abdominal distention develops, accompanied by stercoraceous vomiting. Death may result from the absorption of enterogenic toxins or from perforation and peritonitis.

Subacute and chronic cases may present a history of repeated seizures of colic and obstipation, with or without vomiting. Pain and tenderness in the region of the affected gut are constant symptoms, and cecal volvulus may be confused with appendicitis. Torsion of the small gut occurs most commonly, and the mass usually presents in the upper left abdomen.

**Prognosis** — Intestinal obstruction is rapidly lethal in early life, and the prognosis depends chiefly upon the degree of enterogenic toxemia and gut viability. Cases operated upon early usually recover; late cases requiring resection seldom survive.

**Treatment** — Although in some instances the volvulus may reduce spontaneously, immediate operation is imperative unless reduction has actually occurred. The latter is evidenced by disappearance of the mass and the passage of flatus and feces. Temporization is extremely hazardous and inexcusable.

If there has been excessive vomiting with loss of fluid and chlorides — dehydration and hypochloremia — a preoperative infusion of 5 per cent glucose in physiologic saline should be administered. Gastric lavage should also precede the anesthesia to prevent the possible aspiration of regurgitated material. A right or left mid rectus incision is made sufficiently ample to readily withdraw the torsioned mass into the wound, saline pads being at hand to prevent evisceration.

A moment's study of the mass will reveal its mechanics and the volvulus is then untwisted. In instances of great distention a temporary ileotomy or colotomy above the obstruction may be necessary to unfold the torsion. If there is a question of gut viability, the intestines should be covered with hot moist pads for five minutes. Improvement in color and a return of peristalsis indicate living bowel. Narrow gray necrotic areas may be infolded; more extensive gangrene requires resection. The abdominal wall is then closed in layers with or without intraperitoneal drainage. Retention sutures of silk worm or dermal are advisable to prevent dehiscence.

**Postoperative Treatment**—Restoration and maintenance of the normal water-saline balance is imperative. This may be accomplished through the administration of adequate amounts of physiologic salt solution with glucose by hypodermoclysis or phleboclysis. High ileostomy is seldom performed; duodenal suctionage through a Levine tube is definitely preferable.

## INTESTINAL OBSTRUCTION

Intestinal obstruction is a secondary disease which is caused by some condition that closes a greater or lesser extent of the intestinal lumen. It may be congenital or acquired, complete or incomplete, acute or chronic, and occur in any part of the large or small gut. (Anorectal atresia is discussed in the section on Malformations of the Rectum and Anus.)

### CONGENITAL STENOSIS OR ATRESIA

Congenital atresia is relatively rare. It occurs most often in the small intestine and generally involves the duodenum. In some instances the occlusion is membranous and in others a fibrous cord replaces the gut. Not infrequently there is a multiplicity of defects. In a case reported by the writer the infant exhibited the following: atresia ani and recti; duodenal replacement by a fibrous cord; absence of the gall bladder; bilateral polycystic kidneys, and double left ureter. (Figs. 202 and 203.)

**Etiology**—The conjectural factors operating during fetal life are numerous: fetal bands and luetic or tuberculous peritonitis, fetal volvulus or intussusception, Meckel's diverticulum, and intestinal ulceration.

**Symptomatology**—High complete obstruction produces symptoms within a few hours of birth, and low obstruction on the second or third day. They comprise vomiting, obstipation and progressive dehydration. Nothing is passed rectally except a few meconium stools and mucus. Distention develops less rapidly than in acquired ileus due to the relative absence of intestinal flora. In duodenal obstruction the distention is wholly epigastric; where it is colonic it is general.

Auscultatory peristalsis is usually present and peristaltic waves may be visible. In incomplete occlusion the symptoms are less severe—moderate distention, occasional vomiting and obstinate constipation.

**Diagnosis**—Esophageal obstruction produces immediate vomiting during feeding and the site of atresia may be readily determined by a catheter or barium roentgenogram. Hypertrophic pyloric stenosis seldom develops during the first week, is generally accompanied by visible peristalsis and a palpable tumor, and the vomitus is bile-free. In low obstruction the vomiting occurs late and is of fecal character. The site of obstruction is usually demonstrable roentgenologically without the ingestion of barium. Ano-rectal atresia is diagnosed by inserting the finger into the anus.

**Prognosis**—In cases of complete atresia death occurs within a week. Incomplete obstruction is often compatible with life.

**Treatment**—Complete atresia demands immediate surgery. Un fortunately but few cases are correctable. In partial obstruction operation becomes indicated if the condition becomes progressively worse or if acute ileus develops. Preoperative and postoperative maintenance of the normal water-saline balance is imperative.

## ACQUIRED INTESTINAL OBSTRUCTION

Acquired obstruction occurs much more frequently than congenital atresia. When the antecedent cause is mechanical the condition is termed *mechanical ileus*; when toxic or neurologic, *paralytic* or *adynamic ileus*; and when due to bowel spasm, *dynamic ileus*. The obstruction may be partial or complete. In the former obstinate constipation occurs and in the latter neither gas nor feces passes the point of constriction. The pathologic zone may be narrow and band-like or involve several feet of gut.

When the vascular competency of the gut is damaged the element of strangulation is added. Although in obstructions producing great distention the capillary circulation is of necessity altered, the term *strangulation* generally implies gross vascular damage. The latter occurs in all lethal obstructions and is frequently accompanied by thrombosis or embolism of the mesenteric vessels.

Irrespective of the causative factor, complete obstruction produces the following: stoppage of the fecal stream; distention of the proximal bowel with gas and fluid; circulatory disturbances within the bowel wall of variable degree from venous stasis to gangrene; and the production of lethal producing enterogenic toxins of the histamine type.

**Etiology**—*A. Mechanical Ileus*—The manifold causes may be of external or internal origin. The former comprise the following: herniation through peritoneal openings; retroperitoneal recesses or

hiati in the mesentery, peritonitis by producing plastic adhesions; peritoneal bands, either congenital or acquired; volvulus; and cicatricial contractions due to syphilis or tuberculosis.

The internal or obturation causes comprise intussusception and foreign bodies. The former is by far the most common agent in early childhood (Refer to Intussusception.) Foreign bodies may consist of inspissated meconium in the new-born, enteroliths, masses of ascarides, articles swallowed or rarely, tumors of the bowel.

*B. Adynamic or Paralytic Ileus.*—The etiologic factor may be neurogenic, toxic or circulatory. The first may occur in spinal cord injuries or severe abdominal trauma. Temporary ileus following laparotomy is a common example of the latter. Toxic types are frequently exhibited in peritonitis, pneumonia and typhoid fever. The vascular supply of the intestinal tract is largely a terminal one and thrombosis or embolism readily produces infarctual destruction with resulting ileus.

*Pathogenesis.*—In acute ileus the element of circulatory damage is usually added to that of obstruction. The latter *per se* causes stoppage of the fecal stream with resulting distention of the proximal segment of the bowel and collapse of the distal portion. The retained-back material consists of gastric, biliary, pancreatic and intestinal secretions, plus a variable amount of ingested food.

Although the bacterial content of the upper intestine is less virulent than the highly toxic flora of the terminal ileum and ascending colon, the organisms multiply with great rapidity irrespective of the site of obstruction. Some are gas-producing, and an added translocation of CO<sub>2</sub> may occur from the venous stasis. The rapid formation of gas is accompanied by excessive secretion from the intestinal mucosa and possibly also from the liver and pancreas. The amount of highly toxic fluid which accumulates above the obstruction is often prodigious.

The back log of fluid and gas excites violent peristalsis and vomiting. Intraluminal pressure ultimately compromises the circulation and necrosis and digestion of the mucosa follow. A further highly virulent toxic substance or substances is thereby added. In the final stage of overdistention and paralysis, bacteria may migrate through the intestinal wall, or perforation may occur. The resulting septic peritonitis is rapidly lethal.

In the early stages of obstruction, the distended proximal intestine retains its glistening normal color, later it becomes glazed and dark bluish from venous engorgement, and with beginning gangrene, black and lusterless. The retained contents change from the normal yellow to a blackish blood-tinged stinking fluid.

Histologically, the gut at first exhibits stretching and congestion. Later the capillaries become engorged from venous stasis. Increasing distention ultimately obstructs the arterial inflow and necrosis



follows first of the mucosa and later the outer coats. With secondary thrombosis gross gangrene of the moist type develops. Bacteriemia does not occur except as an agonal complication.

**Toxemia**—In the absence of strangulation pathologic changes occur more rapidly in proximal than in distal obstructions. *In general however the toxemia parallels the degree of damage to the gut wall.* The production and exact nature of the specific toxic substances is incompletely understood. In complete obstruction especially when high there occurs a concentration of the blood with an increase of non protein nitrogenous elements a fall in blood chlorides and progressive alkalosis. Although these findings are explainable on the basis of dehydration with consequent failure of renal excretion the loss of chlorides through vomiting and the loss of electrolytes into the accumulated fluid within the bowel certain highly toxic substances in the nature of histamines or a protease also appear to be produced in conditions of strangulation their absorption is rapidly lethal.

**Chronic Incomplete Obstruction**—In chronic types of partial obstruction the pathologic changes are quite different from those occurring in acute ileus. The fecal material which dams back from time to time above the point of constriction is unaccompanied by any great production of gas or excessive accumulation of toxic fluid. The back log usually undergoes liquefaction and passes through the narrowed lumen.

The dominant lesion is a compensatory hypertrophy of a short segment of gut proximal to the obstruction. This results from continued hyperperistalsis and in many instances there is associated dilatation. In severe cases the mucosa ultimately becomes damaged and the resulting infection and edema of the gut wall may precipitate acute complete obstruction.

**Symptomatology of Acute Intestinal Obstruction** The outstanding symptoms are definite and remarkably constant pain nausea and vomiting stoppage of both fecal stream and flatus abdominal distention general toxemia with increasing rapid feeble pulse and clear sensorium. *Fever and leukocytosis are absent except in the presence of circulatory damage or peritonitis.* In mechanical ileus visible and auscultatory peristalsis may be present whereas in paralytic types the abdomen is conspicuously silent.

**Pain**—In mechanical ileus pain is a constant factor. It is of proximal character and varies in degree from mild cramps to excruciating colic depending upon the acuity of onset completeness of obstruction and associated circulatory damage. Although the pain at first is often general or periumbilical it may later become localized at the site of obstruction.

In beginning strangulation the pain is acutely severe and colicky—the pain of dying tissue. With the advent of gangrene it promptly

disappears—the silence of dead tissue. The sudden cessation of severe pain with persistence of obstruction is thus an ominous sign. In paralytic ileus there is an absence of peristalsis and the pain is of constant rather than colicky type and is generally less severe.

**Nausea and Vomiting** Nausea is commonly present at the onset and at times persists. ushering in the attack the early vomitus consists of stomach contents. After a free interval of a few to several hours emesis recurs and the vomitus is composed of bile, pancreatic secretions and upper intestinal contents. Later it becomes dark and blackish with foul odor and ultimately fecaloid. The early vomiting is generally forceful and projectile later from paralysis of the overdistended stomach it assumes an overflow type and small quantities of stinking coffee-ground fluid are repeatedly spilled without effort. A tremendous amount of fluid may be lost in this backwash vomiting and the resulting dehydration and hypochloremia become acutely severe.

**Obstipation** Fecal stoppage is absolute in complete obstruction. Occasionally one or more stools are passed from residual material below the obstruction or feces are returned in the first enema. Such findings may be misleading. Once the lower bowel is cleansed however further flatus and feces are wanting. In intussusception and certain thrombotic conditions bloody mucus may be evacuated.

**Distention** Abdominal distention usually develops rapidly, especially in infants. In mechanical obstruction of the small intestine the distention occurs chiefly in the central portion of the abdomen and visible or auscultatory peristalsis is frequently demonstrable. In paralytic ileus the distention is generalized and more pronounced and the abdomen is silent.

**Toxemia**—The development of toxemia is one of the most characteristic symptoms of intestinal obstruction. Although it occurs irrespective of the etiologic factor its degree is definitely more pronounced when accompanied by strangulation. Through absorption of enterogenic toxins the pulse becomes progressively more rapid and thready, blood pressure falls and prostration is marked. Dehydration is evidenced early by the pinched expression, sunken eyes, parched and fissured lips and tongue and putty like inelasticity of the skin. (Refer to Dehydration.) The sensorium however remains clear.

**Physical Examination** *Inspection*—The type of distention should be noted whether generalized or limited to the central portion of the abdomen, also the character of the respirations. In mechanical obstructions the breathing is both thoracic and abdominal whereas in peritonitic paralytic ileus the abdomen is splinted and quiet. Visible peristalsis denotes mechanical obstruction.

*Palpation*—Except in paralytic ileus associated with peritonitis the distended abdomen is non-tender and muscle spasm and rebound

tenderness are absent. A mass may be demonstrable in intussusception, pyloric stenosis, volvulus and irreducible inguinal, femoral or umbilical hernia.

**Percussion**—A dull area may indicate the site of the pathology and dulness over the zone of the ascending colon generally denotes intussusception. Early focal tympany is suggestive of volvulus and ascites of tuberculous peritonitis. In advanced cases general tympanites develops and the liver dulness is pushed upward.

**Auscultation**—Swishing, gurgling sounds and borborvgmi are commonly exhibited in mechanical obstruction and may focalize the site of the pathology. In paralytic ileus the abdomen is conspicuously silent.

**Pulse and Temperature**—The character and rapidity of the pulse are valuable indices of toxemic severity. A progressively rising pulse rate is ominous. Intestinal obstruction *per se* is practically an afebrile condition; elevation of temperature occurs mainly with circulatory damage or peritoneal invasion.

**Blood**—In the absence of strangulation, thrombosis or inflammation, the leukocyte count remains normal. The blood exhibits a marked decrease in the non-protein nitrogen, especially of the urea; a diminution of chlorides and an increased CO combining power. Erythrocytosis denotes dehydration.

**Diagnosis**—The cardinal symptom is acute and persistent obstruction of the bowels. In cases presenting a previous history of appendicitis or hernia the diagnosis is usually readily made. In many instances, however, the onset of acute obstruction occurs without demonstrable cause: intussusception, volvulus, constriction from congenital bands and peritoneal recesses, Meckel's diverticulum, etc. In such cases careful evaluation of the sequential symptomatology is of the utmost importance.

The development of acute ileus in diffuse peritonitis is generally assumed to be of paralytic origin. Not infrequently, however, by reason of fibrinous adhesions, actual mechanical ileus occurs and many peritonitis deaths are due to the absorption of enterogenic toxins engendered by obstruction rather than to sepsis. Toxic paralytic ileus usually develops in the first few days following operation; distention is generalized and the abdomen is conspicuously silent. Mechanical obstruction occurs chiefly during the second or third week; the distention is less general, the flanks are seldom involved and auscultatory peristalsis is often evidenced.

**Roentgen Examination**—This may be of definite value in obscure cases of obstruction and flat plates combined with stereoscopic examination may define the site of pathology. Barium should not be administered as it may precipitate complete occlusion in threatened obstruction.

**Course and Prognosis**—The operative mortality is greatest in cases of high obstruction and in those accompanied by strangulation. Most deaths are directly due to the toxemia although peritonitis may be an associated factor. Unrelieved cases of acute obstruction succumb within four or five days.

Cases of subacute or chronic partial obstruction pursue a variable course. While some improve and remain well many become progressively worse and finally develop acute obstruction. Recurrent symptoms warrant surgical intervention.

**Prophylaxis**—Hernia should receive appropriate supportive or surgical treatment. The latter is definitely indicated in all irreducible types. (Refer to Hernia.) Postoperative bands and adhesions which commonly cause obstruction are produced by many factors: peritoneal damage through rough handling, evisceration, prolonged exposure, sponging and rubbing with dry gauze, the use of irritating solutions, and especially the injudicious and prolonged usage of drains. The meticulous gentle handling of peritoneal tissues cannot be overemphasized, also the necessity of infolding or peritonealizing raw serous surfaces.

**Treatment**—Acute intestinal obstruction is seldom relieved by palliative measures. Unless successful immediate surgical intervention is imperative as procrastination of even a few hours may lead to lethal toxemia. The objectives comprise the following: (1) Relief of the obstruction, (2) restoration of luminal patency, (3) evacuation of the toxic bowel content, and (4) restoration of the normal saline-water balance through the administration of supportive measures which combat dehydration, hypochloremia and alkalosis.

In conditions of anhydremia and chloride deficiency, the preoperative administration of glucose in physiologic saline solution through phlebotomy or hypodermoclysis is of inestimable value. (Refer to Dehydration.) Gastric lavage should also precede the anesthetic to prevent the possible aspiration of toxic vomitus. Ether narcosis is generally employed; ethylene, acetylene and cyclopropane are less toxic but may fail to produce sufficient muscular relaxation. In prepubescent children spinal anesthesia may be elected.

**Surgical Approach**—A history of the sequential symptomatology combined with a careful physical examination will usually determine the site of obstruction. The surgical approach is planned accordingly. In obscure obstructions an exploratory mid right rectus incision is advisable as most pathologies are small intestinal, at or near the ileocecal junction (appendicitis, intussusception, Meckel's diverticulum).

The incision, with its center at the umbilicus, is made 1 to 2 cm. to the right of the mid line. After the rectus sheath is opened, the muscle is separated from its attachment to the linea alba and

retracted outward, the posterior sheath and peritoneum are then carefully opened between artery clamps, during inspiration. Adequate exposure is imperative and this approach readily lends itself to enlargement upward or downward. Saline pads should be at hand to prevent evisceration of the overdistended bowel. In mechanical ileus due to peritoneal bands or omental adhesions, severance of the same will relieve the obstruction, following which the back log of fluid and gas passes rapidly beyond. (The treatment of Intussusception and Volvulus is discussed elsewhere.)

In complicated cases and those with great distention, evisceration may be necessary to determine the pathology. The intestines should be carefully received upon, and covered by, warm saline pads or rubber dam, prolonged exposure to the air produces shock and peritoneal damage through chilling and drying. In rare instances a temporary ileostomy may be required to reduce the distention.

**Management of the Obstructed Gut**—After the obstruction has been relieved, the next step is to determine the viability of the gut. If the intestine appears engorged and dark blue with multiple petechiæ, the coils should be wrapped in warm saline pads for five minutes. Improvement in color accompanied by a return of peristalsis indicates viability. Not infrequently a gray band is present, representing the site of lincal constriction. Such bowel tissue is necrotic and, if left *in situ*, will lead to perforation and septic peritonitis. When the band is narrow it may be inverted by circumferential approximation of the healthy gut margins, the suture line being covered by an anchorage of omentum. Wider gray zones, and massive gangrene, require resection. Closure of both ostia and side-to-side anastomosis is simpler and safer than end to end approximation. In certain types of colonic obstruction, a two-stage Mikulicz resection may be elected.

**Drainage**—The question of intraperitoneal drainage following resection is controversial. Most surgeons favor drainage to the vicinity of, but not in contact with, the anastomosis. In cases complicated by septic peritonitis, supportive treatment in the Fowler posture offers the best prospect for recovery. (Refer to Peritonitis.)

In closure of the abdominal wall, the posterior rectus sheath and peritoneum should be meticulously approximated with No. 1 plain catgut, the muscle and anterior sheath with No. 1 chromic, and the skin with dermal or silk. Retention sutures of non-absorbable material are also advisable. The abdominal dressings should be held firmly with wide adhesive straps, supplemented with a scultitis binder. In clean cases the skin sutures may be removed on the eighth to tenth day but the retentions should be allowed to remain.

for at least two weeks. Such prolonged support is advisable to prevent possible dehiscence.

**Postoperative Treatment**—Many otherwise hopeless cases are salvaged by appropriate postoperative treatment. The problem comprises (1) Measures to combat dehydration, hypochloremia and alkalosis; (2) evacuation of the back log of septic material within the gut; and (3) restoration of peristalsis.

The first is specifically combated through the administration of physiologic saline solution by repeated phlebotomies or hypodermocentesis in amounts sufficient to restore the normal saline-water balance. The value of such supportive therapy cannot be overstressed. (Laboratory dogs with high intestinal obstruction die in three or four days; however, if saline is injected under the skin in sufficient quantities to offset the loss by vomiting, life may be sustained for weeks.) The injections of saline solution should be continued until the oral intake suffices. The addition of 5 per cent glucose solution is definitely valuable in addition to supplying a readily oxidizable food; it combats alkalosis and stimulates renal output. Fluid by proctocentesis is messy and impractical. (For saline dosage refer to the chapter on Dehydration.) In critical cases, blood transfusion is of great value.

The operation of jejunostomy or high ileostomy, formerly employed for drainage of the back log of septic fluid, has been largely superseded by duodenal suction drainage through the indwelling Levine catheter passed intranasally. Repeated colonic irrigations empty the bowels from below.

Turpentine stupes stimulate peristalsis and are most effective when followed by prolonged warm colonic irrigations. The latter may be preceded by the hypodermic injection of eserine sulphate or surgical pituitrin. Opium and morphine should be withheld in cases of severe pain; small hypodermic doses of codeine are definitely less harmful. With the return of moisture to the tongue, the passage of gas and feces, and the subsidence of distention, oral fluid intake may be started.

A small number of cases have repeated attacks of acute complete obstruction which necessitate multiple laparotomies. The underlying pathology is chronic plastic peritonitis and in certain instances careful exploration will reveal tuberculosis of the Fallopian tubes, cecum or appendix. The removal of such primary nidus generally prevents recurrence. In non-tuberculous plastic peritonitis, the intraperitoneal installation of amniotic fluid appears to be of value.

## CHAPTER XXX

### APPENDICITIS

#### ACUTE APPENDICITIS

ALTHOUGH within very definite limitations there should be no mortality from acute appendicitis the death rate remains appallingly high. This is largely attributable to lay ignorance or professional procrastination. In the presence of cramps the unwitting mother often administers a cathartic especially the death producing lullaby of castor oil and the over hopeful physician may delay surgical council until peritonitis develops.

**Anatomy**—The appendix varies greatly in size shape and position. Usually 7 to 10 cm. its length may vary from 1 cm. to over 20 cm. Relatively large at birth it tends to undergo atrophy and obliteration after middle life. Absence of the organ is exceedingly rare and reduplication has been observed.

The appendix is situated on the postero inner side of the caput coli at the end of the anterior tenia (an excellent guide in locating it). Although normally occupying the right lower quadrant through arrest or failure of colonic rotation it may lie under the liver spleen in the left lower quadrant or at any intermediate point (Fig 169). It may also be found in the pelvis in the site of an inguinal femoral or umbilical hernia and in exceedingly rare cases the organ may be inverted. Its situs in the right lower quadrant is also subject to considerable variation (Fig 170). Diverticulosis is observed very rarely.

The ileocecal valve is a physiologic partition point between the relative stasis and alkalinity of the terminal ileum and the marked fluidity and mild acidity of the cecum. Whereas the intestinal contents of the latter exhibit the highest and most varied number of virulent bacteria the sphincteric zone is well fortified by abundant protective lymphoid tissue.

**Luminal Variations**—The appendix of infants and young children differs from the adult type in several anatomic respects. The lumen of the infantile organ tends to be funnel shaped and increases in diameter from tip to base. Adequate drainage afforded thereby accounts for the rarity of appendicitis in infants under two years of age. During later childhood the lumen becomes small tubular and of relatively the same diameter throughout its length and the lymphoid follicles may encroach upon it. Both conditions are

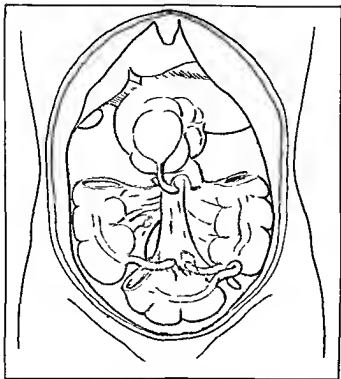


FIG 169 — Variable locations of the cecum and appendix due to a long cecal mesentery or to incomplete colonic rotation and descent (After Kelly)

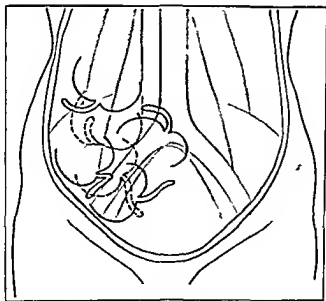


FIG 170 — Common variations in the location of the cecum and appendix in the right lower abdomen (After Kelly)



favorable to stasis, obstruction, the multiplicity of organisms and bacterial invasion of the submucosa

**Gerlach's Valve** or guard-like fold of mucous membrane at the junction of the appendix and bowel is relatively poorly developed in childhood and offers little protection. Moreover, the muscular walls of the organ are relatively thin, the submucosal tissue being especially scant in comparison to the fibrous inelastic structure present in the adult type. Thus paucity of a submucous layer and the presence of a wide lumen account for the lack of sequential symptomatology in the acute appendicitides of young children. Although there is greater tendency to intracecal drainage, the lack of a firm submucous layer predisposes to early perforation.

The **Appendicular Artery** supplying the appendix is a terminal vessel in approximately 88 per cent of cases. Thrombosis of the main trunk produces gangrene of the entire organ, and that of a branch, focal gangrene (infarct). The deficiency of serosa at the mesenteric border represents the weakest luminal point.

**Predisposing Causes** — *Age* — Although occurring at all ages, appendicitis is most common in the "teens". It is infrequent under five years of age (not more than 3 per cent of all cases) and rare under two years. (Gloniger successfully operated upon an infant forty-one hours old.)

*Sex* — The incidence is higher in males.

*Familial* — Several members of a family may be afflicted either through faulty diet or common anatomic peculiarities favorable to infection.

*Diet* — Constipation or diarrhea due to dietary indiscretion is the most frequent predisposing cause.

*Trauma* — Overexertion may reduce resistance to infection and excessive intra-abdominal pressure may also be a factor. Direct trauma may incite a preexisting latent infection.

*Previous Attacks* — One attack predisposes to recurrence, usually within three months.

*Infectious Diseases* — Appendicitis frequently follows infections that produce lymphoid hyperplasia: tonsillitis, scarlet fever, measles, acute respiratory diseases and especially enteritis.

*Phylogenetic*. — Being a vestigial organ without function, the appendix is predisposed to inflammation through functional inactivity.

*Anatomic Factors* — The appendix occupies a relatively high position in infancy and descends into the right lower quadrant between the third and sixth year. It normally dangles loosely from the postero-inner side of the caput coli in a downward and inward direction. The position of the organ is subject to wide variation, however, and it may lie in any axis — in front of or behind the cecum, to its inner or outer side, or behind the peritoneum (the result of incomplete descent). It may be kinked, due to a short mesentery or

angulated from scar tissue bands. Any of these factors may hinder physiologic emptying and the resulting stasis and circulatory interference predispose to infection. Inflammation of the organ may result in fibrotic obliteration of its lumen. When this occurs in the proximal portion, the distal lumen may become shut off and through continued secretion produce a cyst or hydrops of the appendix. Adherence of the tip of the appendix to adjacent viscera may also interfere with adequate drainage.

**Tumors**—Benign tumors are of rare occurrence and comprise polypus, fibroma, myoma, myxoma and lipoma. Carcinoma is almost always associated with adult life and the growths are usually localized within the organ. They are rarely malignant and appendectomy results in cure.

**Exciting Causes**—*Foreign Bodies*—Fecal concretions, termed fecoliths, enteroliths or coproliths, occur in over 30 per cent of cases and are definite exciting causes of appendicitis. Their presence is indicative of previous mild catarrhal inflammation. The latter interferes with adequate drainage and the retained inspissated mucus becomes deposited about clumps of bacteria or small fecal particles, thereby producing concretions. (A similar genesis to that of gall stones.) The enteroliths occasionally become encrusted with lime salts and cast roentgen ray shadows resembling ureteral calculi. Large concretions may produce decubitus necrosis resulting in secondary bacterial invasion.

**Intestinal Parasites**—*Oxyuris* are often found in the appendix and are probably inconsequential. *Ascaris lumbricoides* occur infrequently and may be a provocative factor. The incidence of exogenous foreign bodies such as berry seeds and small metallic objects has been much exaggerated. They play a very minor etiologic rôle.

**Bacteriology**—In order of frequency of cultural growth, the following organisms are associated with acute appendicitis: *B. coli*, staphylococcus, streptococcus, *B. proteus*, *B. tuberculosis*, pneumococcus, streptothrix, actinomycosis and numerous anaerobic organisms. However, there is considerable controversy concerning the primary and secondary invaders and many believe that the streptococcus is the sole, or dominant, primary organism.

Failure to distinguish between primary and secondary invaders accounts for the wide variation in statistical reports. When bacteriologic data is compiled on the basis of six, twelve, twenty-four and forty-eight hour appendicitides, streptococci predominate in the majority of early cases, and after twelve to twenty-four hours, *B. coli* are most common.

The staphylococcus so frequently present with colon bacillus infections appears to occupy an intermediate position in the production of peritonitis and is probably a secondary protective, rather

than a primary causative agent. The organisms are usually present in great number on the outskirts of the inflammatory zone and act as a defense mechanism by creating delimiting adhesions.

*B. coli* pus is readily recognized by its characteristic foul odor. *B. pyocyaneus*, a normal inhabitant of the intestine, produces green pus only in the presence of oxygen which is necessary for the production of pyocyanin pigment. Although capable of producing a virulent peritonitis, infection therefrom is usually localized. The organism is never associated with the colon bacillus. (Refer to Peritonitis.)

**Pathology**—Inflammation of the appendix may be acute, subacute, or chronic. The various varieties are mere gradations of the same pathologic process. The chronic type always results from previous acute inflammation of greater or lesser degree.

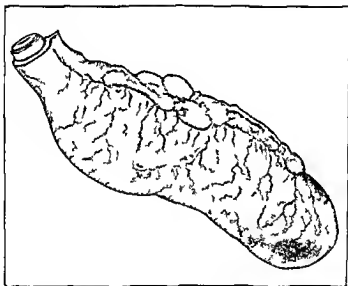


FIG. 171.—Mucocoele or cyst of the appendix from stricture of the proximal end of the lumen.

**Acute Catarrhal Appendicitis** In the simplest form of inflammation there is congestion, edema, and round cell inflammation of the mucous membrane, and especially of the lymph follicles. The appendix becomes thickened and distended from the retained mucus, and the surface veins are congested. If the obstruction be relieved, complete resolution may follow. More often, however, there is some inflammatory residuum evidenced by more or less constriction of the lumen. Occasionally a proximal obstruction persists, and the accumulating mucus produces a mucocoele or appendicular cyst (Fig. 171). If the inflammation has been more penetrating, adhesions may result from the protective fibroplastic exudate.

Although the mildest type of catarrhal inflammation may be accompanied by only slight digestive disturbance such repeated and apparently inconsequential invasions may eventuate in diffuse fibrous infiltration chronic appendicitis. The appendix becomes firm and thickened often with constrictions at one or more points and adhesions may connect it with adjacent structures. Acute inflammation may develop at any time.

**Acute Diffuse Appendicitis**—In other instances the inflammation is more severe and the submucous muscular and peritoneal coats become progressively involved. The inflammation may be limited to a segment containing a concretion or invade the entire organ. The appendix becomes red swollen and tense often covered with fibrin and a zone of clear or slightly turbid exudate may surround it. The process may resolve and result in chronic diffuse appendicitis or progress to suppuration and the pus be discharged into the lumen—acute suppurative appendicitis.

When the process is more severe greenish or black areas of necrosis appear resulting in perforative appendicitis. With more intense inflammation thrombosis occurs and depending upon whether the thrombosis involves a branch of the appendicular artery or the main trunk a segment of the appendix or the entire organ becomes grayish green mushy and gangrenous—gangrenous appendicitis.

Severe infections are accompanied by involvement of the peritoneum. Although this generally results from perforation or gangrene bacterial invasion may occur without a macroscopic break in continuity. In certain types an abundant fibrinous exudate produces agglutination of the neighboring intestinal coils and omentum and the purulent process becomes localized—appendiceal abscess. This commonly occurs with *B. coli* and staphylococcus infections. In the presence of virulent streptococcus invasion the exudate is thin scant and low in fibrin content and diffuse peritonitis results.

**Operative Findings**—The usual findings at operation in acute infections of twenty four to thirty six hours duration are as follows: the vessels of the abdominal wall are congested and bleed freely upon opening the edematous peritoneum clear fluid is encountered and as the region of the appendix is approached an increasing amount of turbid fluid or frank pus appears often with the characteristic foul odor of *B. coli* infection. The peritoneum of the adjacent intestinal coils and omentum is acutely reddened covered with fibrin and agglutinated about the appendix. At this stage and especially in young children in whom the omentum is short there is frequently a considerable amount of turbid fluid in the pelvis which may later eventuate in a pelvic abscess unless evacuated.

After several days an unrelied appendiceal or pelvic abscess may rupture into the peritoneal cavity and produce fulminant spreading

peritonitis in unusual instances the pus is evacuated into the intestine through ulceration of its wall. At any stage the appendix may slough and lie free in the abscess.

In severe streptococcus infection of twenty four to thirty six hours duration the operative findings exhibit a marked subperitoneal cellulitis and upon opening the abdomen there is little or no exudate and no odor. The peritoneum appears blistered lustreless thickened edematous and hyperemic even with petechiae. Fibrin and delimiting adhesions are wanting and the inflammation is widespread and diffuse. Such lethal types are fortunately uncommon.

**Chronic Appendicitis.** The appendix is endowed with remarkable reparative potential. This is evidenced by the crippling scars and dense adhesions so frequently exhibited. The mesentery may be thickened and shortened and the appendix stubbed and widened. Adhesions may cause a multiplicity of sharp angulations and kinks and at times the organ is completely buried in a mass of scar tissue. Occasionally the chronic inflammatory process spreads to the cecum and produces a thickened brawny mass which suggests tuberculosis.

Histologically scar tissue dominates the pathology and the fibrosis is especially conspicuous beneath the mucosa. Such scarring definitely inhibits normal peristaltic expulsive forces and enteroliths are formed frequently. Their presence is a common cause of appendicular colic. Although progressive fibrotic obliteration of the lumen is a physiologic process in middle life the presence of scar tissue obliteration and atrophy in childhood is definite evidence of previous inflammation. *In an appendix once damaged recurrence of infection is the rule.*

**Primary Tuberculosis** of the appendix is rare in children and the pathology is usually associated with tuberculous peritonitis. In the very unusual cases of *actinomyces* the organ becomes involved through ingestion of the fungus. A large brawny induration develops which usually involves the cecum. Suppuration follows with the formation of multiple sinuses. The ray fungus is readily demonstrable in the sulphur like granules extruded in the discharge.

### Symptomatology of Acute Appendicitis

Although the sequence of symptomatology in children under twelve years of age may be somewhat modified due to (1) the paucity of a resistant submucosa and (2) a greater tendency to intracecal drainage the clinical picture is generally definite and decisive. Infection almost always begins in the mucosa and the products of inflammation are retained within the lumen by the swollen mucous membrane. Exaggerated but ineffective expulsive peristalsis produces (1) colicky pain which occurs suddenly is

severe and recurrent in character and usually reaches its maximum in about four hours. This primary pain due to violent peristalsis above the provoking cause is referred to the epigastrium. Its onset is accompanied by (2) nausea with or without vomiting. Unless a cathartic has been administered the nausea and vomiting soon subside. (Recurrence of vomiting after twenty-four hours suggests peritonitis.) The abdomen is (3) generally sensitive but seldom spastic in the first hours. Rectal temperature reveals (4) slight fever. With rare exception the onset is paralleled by some degree of (5) leukocytosis and polymucocytosis. This sequence of pain, nausea, abdominal tenderness, fever and leukocytosis so admirably first emphasized by Murphy is definite and constant. The infection may eventuate as follows: (1) by intracecal drainage and resolution; (2) suppuration and perforation; or (3) gangrene.

**Focalization in the Right Lower Quadrant**—With involvement of the serosa which usually occurs in four to six hours the abdominal symptomatology changes to that of a focal pathologic process in the region of the appendix. The pain becomes constant and localized in the right lower quadrant and nausea and vomiting cease; the general abdominal tenderness changes to a local point of tenderness capable of accurate focalization at McBurney's point (appendicular base) and muscle spasm is usually demonstrable. The temperature may temporarily subside but the leukocytosis and polymucocytosis persist or increase. At this stage the disease is diagnosed as acute appendicitis although the pathologic process is actually an incipient localized peritonitis (serosal inflammation).

Peritoneal response to bacterial invasion is immediate and specific: first a transudate then an exudate is thrown out and the neighboring intestinal coils and omentum become agglutinated about the process. With fixation of the intestines peristalsis ceases and distention develops. At this stage a vigorous cathartic is often given and the localized peritonitis becomes converted into a process of rapidly ascending pathologic severity—that of spreading diffuse peritonitis.

**The Danger of Catharsis**—The danger of catharsis in appendicitis is forcefully emphasized by the statistics of Heald. Of 402 patients of all ages who had appendicitis but did not take a laxative, 1 out of 80 died; of 992 who took one laxative, 1 out of 14 died; and among 992 who took two or more laxatives, 1 out of 7 died.

**Pain**—The onset of appendicitis is not always immediately accompanied by colicky pain. Occasionally and especially in young children there may be an upset stomach for a few days with only vague abdominal discomfort. In such cases the epigastric pain may take several hours to develop acutely. If there has been a previous attack the pain may be felt in the right lower quadrant rather than in the epigastrium.

The secondary localization of pain (serosal inflammation) varies with the site of the appendix. Pain over the iliac crest extending into the loin generally denotes a retrocecal appendix lying to the outer side of the cecum. When the organ is directed upward and inward the pain may focalize near the umbilicus. If the appendix lies entirely within the pelvis the abdominal signs may be negligible. Increased frequency and urgency of urination are suggestive symptoms. To illustrate: A colleague's son aged ten years came home from school complaining of nausea and abdominal pain. Examination by the father was negative except for slight abdominal tenderness. Mouth temperature was normal. An enema which temporarily increased the cramps was followed by relief. At bedtime 6 ounces of citrate of magnesia was administered. During the night the patient was awakened by recurrent cramps and toward morning developed frequent micturition. Seen twenty six hours after the onset the rectal temperature was  $100.4^{\circ}\text{F}$  and pulse 80. The abdominal examination failed to reveal either tenderness or muscle spasm. Rectal examination however elicited acute tenderness on the right side. The blood exhibited a leukocytosis of  $12,100$  with polynucleosis of 81 per cent. Immediate operation revealed a perforated intrapelvic appendix surrounded by several ounces of turbid fluid with B. coli odor. Convalescence was uneventful.

**Vomiting**—As a rule the vomiting is not protracted and generally occurs only once or twice unless medication has been administered. Recurrence of vomiting after twenty four hours suggests peritonitis. In the late stages of peritonitis the progressive distention from paralytic ileus is often accompanied by repeated copious vomiting without effort. Toxic diarrhea may occur with pneumococcus or streptococcus peritonitis.

**Temperature** Rectal temperature in the early stages varies from  $100^{\circ}$  to  $101^{\circ}\text{F}$  and at the end of twenty four hours may reach  $103^{\circ}\text{F}$ . It is seldom higher except in fulminating or severe retrocecal appendicitis. The degree of fever however is no reliable index of the severity or acuity of the process; a normal temperature may even occur with a gangrenous appendix and in rare instances with perforation. Sudden increase in temperature suggests spreading infection and its gradual reduction limitation or subsidence of the process.

**Suppurative Appendicitis** The constitutional symptoms of suppurative appendicitis depend chiefly upon the degree of septic absorption. Although the temperature is usually  $102^{\circ}\text{F}$  plus it may remain low. As previously stated the degree of fever is an unreliable and misleading indicator of the pathology. The pulse is generally rapid and focal tenderness is often acute. The most exquisite tenderness occurs in cases where the appendix lies in con-

tact with the peritoneum of the anterior abdominal wall or when a cyst of the appendix becomes infected. It should be emphasized that muscular rigidity in infants and young children is decidedly less marked than in adults. At times it may be exceedingly slight even in the presence of diffuse peritonitis.

**Development of a Mass**—Considering the paucity of abdominal symptoms in some cases the development of an inflammatory mass is often surprising. After a few days of anorexia and nausea perhaps with slight fever and indefinite cramps and following the administration of laxatives a mass develops in the right lower quadrant. At operation these cases generally present a localized peritonitis with abundant plastic exudate but without gross evidence of perforation. Infrequently a perforation with frank pus obtains.

**Perforative Appendicitis**—With the advent of perforation the pain is often ameliorated through pressure relief of the intra appendiceal empyema and the patient feels better. This critical period of intraperitoneal invasion may be mistaken for improvement until the process eventuates in abscess formation or diffuse peritonitis. The latter is especially apt to occur in young children for in early life the frail omentum is a poorly developed defense mechanism. Severe pain after primary subsidence is ominous and usually implies beginning peritonitis.

**Gangrenous Appendicitis**—A positive diagnosis of focal or total necrosis of the appendix is often impossible. The outstanding features of the pathology are sudden cessation of acute pain and the persistence of acute focal tenderness over McBurney's point. The temperature is notoriously unreliable and may be normal or only slightly elevated. The pulse however is usually accelerated and the blood count exhibits some degree of absolute and relative polynucleosis. Sudden cessation of pain with persistence of acute focal tenderness is a definite indication for immediate operation.

**Spreading or Diffuse Peritonitis**—It should be emphasized that pathologic processes advance with great rapidity in early life and that appendiceal perforation often occurs within twelve hours. Fortunately the protective potential of the peritoneum is maximal in childhood and the majority of perforations become walled off and result in inflammatory masses or frank abscesses.

Of the several factors predisposing to diffuse peritonitis the administration of a cathartic is predominantly the chief cause. Through the incitement of violent peristalsis an intra appendiceal empyema may readily become ruptured and produce massive invasion of the peritoneal cavity. In virulent streptococcus types a minimum of protective fibroplastic exudate is produced and the infection spreads by both surface extension and via the subserous



lymphatics The patient becomes overwhelmed by the intense toxemia before there is time for peritoneal delimitation

Occasionally a chill occurs at the time of peritoneal invasion and there may be reflex vomiting The temperature often rises and the pulse becomes accelerated Spreading tenderness, however, is the most valuable sign of diffusing peritonitis Muscular rigidity is unreliable and may be wanting in young children More often hypersensitiveness of the skin may be elicited In older patients, a head zone of hyperesthesia may be demonstrable Rectal tenderness, especially on the right side, is also an important early diagnostic sign With the gloved finger well lubricated and gently insinuated into the rectum, the small pelvis of the child may be palpated with little discomfort

**Blood Examination**—Although leukocytosis is a relatively constant factor in acute appendicitis, it is subject to wide variations It should be remembered that in children under six years of age, the normal leukocyte count approximates 9500 per c mm, and that in infancy the proportion of lymphocytes is 50 to 60 per cent and the polymorphonuclear neutrophils, 30 to 40 per cent These differentials gradually alter and approach the adult type at eight or ten years

A leukocytosis of 12,000 to 20,000 appears early and is characterized by a relatively high proportion of polymorphonuclear neutrophils This increase in the white blood cells is a reaction to absorption and a moderately high count generally indicates good resistance Slight leukocytosis is equivocal it may occur in mild catarrhal appendicitides or in fulminating infections with overwhelming toxemia Whereas a diminishing leukocytosis in the presence of general improvement is a favorable indication, a low count, with evidence of diffusing tenderness, is ominous The Schilling index test is also of considerable value (Refer to Peritonitis) Although in exceptional cases the leukocytes may number 30,000, a count above 25,000 is suspicious of pulmonary infection

**Diagnosis**—Whereas typical cases of acute appendicitis can often be diagnosed over the telephone atypical types may be very perplexing, especially in young children The history and sequence of symptomatology are of the greatest importance Much can also be gained by observation a child with peritoneal irritation lies quietly on his back or side, with thighs elevated and knees flexed, avoiding any movement, pressure on the abdomen or deep breathing Tenderness and hyperesthesia of the skin are often more evidenced in young children than reflex muscle spasm, in older patients the physical signs are the same as in adults

In early cases, local tenderness is often increased by contraction of the psoas muscle upon flexing the thigh with the leg extended steady pressure made over McBurney's point intensifies the pain

Rebound tenderness is a definite sign of peritoneal involvement and its importance cannot be overstressed. Repeated examinations at hourly intervals are often of great value. If doubt exists after careful observation exploratory laparotomy is less hazardous than procrastination.

**Differential Diagnosis — Pneumonia and Pleurisy** — Pleural pain may be referred through the intercostal and abdominal nerves to the region of the appendix. The associated abdominal tenderness however is superficial and the muscle spasm is relaxed during inspiration. Moreover the temperature, respiratory rate and leukocyte count are usually higher than in appendicitis. In cases of doubt a chest roentgenogram is often diagnostic. In unusual instances acute appendicitis may occur simultaneously with pneumonia.

**Pyelitis** — Pain in acute pyelitis may be referred to the right lower quadrant. The condition is often associated with tenderness over the kidneys which may be elicited by deep palpation or percussion of the flank area. Clumps of leukocytes are usually demonstrable in the urine.

**Gravel** — The passage of fine gravel may produce ureteral colic simulating appendicitis. Fever abdominal tenderness and muscle spasm are absent and the urine generally exhibits crystals and red blood cells.

**Intestinal Colic** — This may cause anxiety especially if there is much fecal accumulation in the colon. Fever leukocytosis and definite signs of appendicular inflammation are wanting and the condition is relieved by enemas.

**Acute Febrile Indigestion** — Differential diagnosis in the first few hours may be impossible. Although the temperature rises rapidly, focal tenderness and muscle spasm do not develop.

**Ileocolitis** — The vomiting fever and abdominal tenderness are associated with frequent diarrheal stools. focal appendicular signs are wanting.

**Influenza** — Although abdominal pain and mild general tenderness may occasionally be associated with influenza focal symptoms are absent and the blood usually exhibits leukopenia.

**Intussusception** — The intermittent colic with free intervals of comfort the development of a mass and the passage of currant jelly stools (blood streaked mucus) are pathognomonic.

**Inflamed Nodes** — At times acute inflammation of the mesenteric or retroperitoneal lymph nodes is associated with acute infections of the throat ears sinuses or poliomyelitis. The abdominal tenderness and rigidity are seldom as focalized or marked as in appendicitis. (Refer to Mesenteric Lymphadenitis.)

**Acute Tuberculous Ileitis** — Acute involvement of the nodes at the ileocecal angle may be confused with appendiceal abscess. High leukocytosis and polynucleosis are absent.

*Psoas Abscess* — A cold abscess resulting from spinal caries may be mistaken for an appendicular mass. The history of chronicity and the spinal roentgenogram readily differentiate the condition.

*Peritonitis* — Unusual primary types (?) due to the pneumococcus or streptococcus may be impossible of differentiation. (Refer to Pneumococcus Peritonitis.)

*Diverticulitis* — Acute inflammation of Meckel's diverticulum or perforation of a diverticular ulcer may closely mimic appendicitis. The focal signs are generally near the umbilicus. (Refer to Meckel's Diverticulum.)

*Volvulus* — The pathology seldom develops in the right lower quadrant. (Refer to Volvulus.)

*Ovarian Cyst or Dermoid* — An ovarian cyst may rarely develop before pubescence and become twisted about its pedicle. Recurrent intermittent colic with the immediate presence of a pelvic mass are diagnostic.

*Persistent Urachus* — In inflammation of a persistent urachus there is a previous history of umbilical discharge with the gradual development of a tender mid line elongated mass extending downward from the umbilicus.

*Torsion of the Omentum* — The condition is exceedingly rare in childhood. There is usually a preceding history of operation or of hernia. The diagnosis is rarely made prior to laparotomy.

*Omentitis* — Except when associated with peritonitis inflammation of the omentum is usually a sequel of omental resection in hernia especially when silk ligatures have been employed.

*Torsion of the Spermatic Cord* (Refer to Chapter XLIII.)

*Acute Cholecystitis and Perforated Ulcer* — The former is very uncommon in childhood and perforation of a gastric or duodenal ulcer is exceedingly rare. A good general rule with children is to consider all acute abdominal inflammations to be of appendiceal origin until proven otherwise.

**Complications of Acute Appendicitis** — The dominant complication of acute appendicitis is peritonitis either local or diffuse. Secondary abscess may result therefrom in the region of the appendix in the pelvis right lumbar gutter left lumbar gutter subdiaphragmatic or subhepatic space or in the soft parts. (Fig 172.)

**Intestinal Obstruction** — Paralytic ileus (adynamic) occurs chiefly during the first few days following operation whereas mechanical obstruction from plastic adhesions generally develops during the second week or later.

**Fecal Fistula** — Fecal fistula may result from sloughing of the cecal wall about the appendix base or from improper treatment of the appendix stump. Most fistulae close spontaneously but may require months for healing.

**Thrombophlebitis** — This may occur by direct extension or through embolism and lead to grave complications in either the systemic or portal system. The clot originates either at the site of the wound or in the pelvic or femoral veins and emboli therefrom lodge most often in the pulmonary vessels. This dramatic catastrophe usually develops toward the end of the second week. The patient suddenly

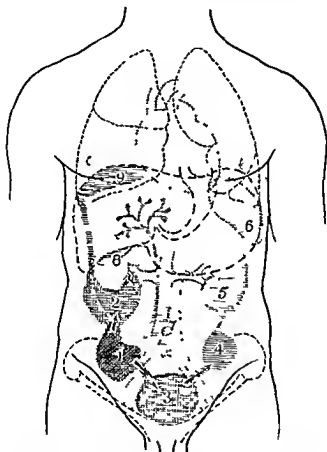


FIG. 172 — Diagrammatic representation of the common sites of primary and secondary appendiceal abscess and usual routes of extension. The more common are shaded the deepest. Suppurative pyelitis is also indicated secondary to portal thrombosis. (After Kelly.)

complains of distress or acute pain in the chest, becomes cyanotic and may succumb in a few minutes if the embolus obstructs a main pulmonary artery. Smaller infarcts are accompanied by pallor, rapid pulse, cough and frothy blood-tinged sputum, and the patient may gradually recover. *Pulmonary embolism* occurs about once in every 2500 appendectomies, the incidence being higher in suppurative than in clean cases.

Thrombophlebitis of the portal system may lead to *pylephlebitis* and the development of multiple abscesses in the liver. There may be a chill at the onset followed by intermittent or remittent fever and sweats. The liver gradually becomes enlarged and tender and slight jaundice is common. The prognosis is extremely grave. (Refer to Pylephlebitis.)

**Pneumonia** — Postoperative pneumonia may develop within forty eight hours following operation. This is usually a lobar type and results from pneumococci harbored in the nasopharynx. During the second week bronchopneumonia may occur from minute septic emboli. The incidence of pneumonia varies from 1 to 3 per cent irrespective of the type of anesthesia employed. *Elective operation should never be performed if the child has a cough or reddened pharynx.*

**Massive Collapse of the Lung** — This unusual complication may occur within the first few days following operation. The patient develops cough, dyspnea, cyanosis and increased pulse rate. Respiratory excursion is definitely limited on the affected side, the percussion note is high pitched, breath sounds absent and the heart may be displaced toward the affected side. Roentgen ray examination is diagnostic. Recovery usually follows in seven to ten days.

**Pyelitis** may develop during any period of the convalescence. **Septicemia** with demonstrable bacteremia is a common complication in severe streptococcus peritonitis.

**Dehiscence** occurs more frequently in children than in adults. It may result from continuous distention, coughing or delirium, improper wound closure especially of the peritoneum or from too early removal of sutures.

**Sequelæ** — The prevention and treatment of postoperative adhesions is discussed elsewhere. (Refer to Postoperative Peritonitis.) **Ventral hernia** may result from division of the motor nerves from prolonged drainage, persistent coughing, continuous distention or excessive fascial sloughing. The incidence is less in drainage cases through a split rectus incision than in the gridiron muscle separation. **Numbness** over the groin or scrotum occasionally occurs from division of sensory fibers. It is not permanent and normal sensation is usually regained within a year.

### CHRONIC APPENDICITIS

The term is rather loosely applied to two groups of cases. In one there is a history of recurrent attacks of acute appendicitis with more or less interval indigestion and focal tenderness. In the other the chief complaint is discomfort or dull pain in the right lower quadrant with or without reflex epigastric symptoms. Although previous acute attacks are denied, careful questioning will usually elicit a history of abdominal cramps at some former

period as chronic appendicitis is always the sequel of antecedent inflammation however mild

Patients with a history of recurrent acute attacks with interval discomfort or focal tenderness are readily diagnosed. Those complaining only of vague epigastric symptoms with tenderness in the appendix region require careful study. Well-developed sthenic children who have repeated indigestion without dietary indiscretions or bowel irregularity and in whom there is definite focalized tenderness over McBurney's point usually have appendiceal pathology. Conversely asthenic types especially girls who are habitually constipated and in whom discomfort and tenderness in the right lower quadrant are the only symptoms generally have visceroprosis or spastic colitis. Such cases are made definitely worse by appendectomy. Imaginary appendicitis occasionally develops in neurotic children when their friends are afflicted.

**The Diagnosis of Chronic Appendicitis**—This is less confusing in children than in adults because the possibility of gall bladder or pyloro-duodenal pathology is negligible. In the absence of previous attacks the most valuable diagnostic findings are the following: acute tenderness definitely focalized at McBurney's point; referred para umbilical pain upon deep pressure over the appendix; nausea induced by sustained deep palpation and a highly tympanitic cecum. The focal pain is often accentuated by proas tension; this may be elicited during palpation by having the patient flex the thigh with the knee held in extension.

**Roentgenologic Examination**—Roentgen ray and fluoroscopic examinations are often of great value in doubtful cases. The presence of enteroliths is indicative of previous inflammation as are also bands, kinks and adhesions. Visualization of the appendix may be absent in obstructive types and a barium residue in the organ after seventy-two hours often denotes pathology. In some cases the shadow of a ureteral calculus or calcified lymph node clarifies the diagnosis.

**Prognosis in Acute Appendicitis**—Most deaths from appendicitis are a sad commentary upon the evils of lay ignorance or professional procrastination for with rare exception the mortality is definitely and solely that of peritonitis (5 to 15 per cent). The danger of interval appendectomy in a surgically competent child is negligible and operation during the first twelve hours of an acute attack almost always eventuates in a smooth and speedy convalescence. With each succeeding hour the danger of perforation or gangrene becomes more ominous. A careful study of statistics reveals an alarmingly progressive incidence of peritonitis from perforation or gangrene after twenty-four hours. Kresch reviewed the appendicitis problem in New York City and in 4049 cases of acute appendicitis operated in fourteen hospitals of high standard the average

mortality was 7 per cent. In the United States the annual roster of martyrs from appendicitis is over 25 000.

**Treatment of Acute Appendicitis** —During the early stages of an acute attack it is often impossible to determine whether the process is catarrhal, suppurative or gangrenous and the only safe procedure is immediate surgical intervention. This is especially true in young children because the symptomatology is less sequential and the danger of diffuse peritonitis is greatest. (Refer to Pathology.) *Early operation by the casual surgeon is a much safer procedure than late appendectomy in the hands of the most skillful.*

Following perforation and in the presence of spreading peritonitis delay is extremely dangerous for localization of the process with resultant abscess formation is definitely less common than in adults. Exception however should be made in cases of fulminating ascending infection of over forty-eight hours duration. In this type supportive treatment with absolute peristaltic rest in the Ochsner posture is preferable to immediate appendectomy and drainage. (Refer to Treatment under Peritonitis.)

Early acute cases with upper respiratory infection should be observed at hourly intervals. If the appendiceal inflammation does not subside operation becomes imperative. The choice of anesthetic and its skillful administration are extremely important. (Refer to chapter on Anesthesia.)

**Treatment of Chronic Appendicitis** —Operation should be advised in cases presenting a history of recurrent attacks even though they be mild in character. A subsequent attack may be fulminant and occur during some intercurrent infection. Operation is also indicated in cases of chronic appendicitis definitely demonstrable by physical or roentgen ray examination. Interval appendectomy is an ideal surgical procedure.

**Preoperative Management** —During an acute attack absolutely nothing should be given by mouth: no food, no water, no medication. Even sips of water are a biologic stimulant of peristalsis.

Freezing the appendix is held in high esteem by the laity. Unquestionably the application of cold over the inflammatory zone tends to diminish peristalsis and is thereby comforting. Unfortunately it masks both the degree of pain and the extent of tenderness and rigidity. Moreover a lowered local resistance to the spread of infection may result for polymorphonuclear leukocytes appears to be reduced by prolonged application of cold to the abdomen.

After operation has been decided upon, codeine or morphine and atropine may be given hypodermically to relieve pain and in early cases a warm soap water or milk and molasses enema is advisable. If dehydration and hypochloremia have developed from excessive vomiting an infusion of 5 per cent glucose in physiologic saline solution should be administered in dosages of 100 to 500 cc. accord-

ing to body weight (Refer to Dehydration) Gastric lavage is best accomplished by siphonage through a Levine tube passed intranasally

The child should be well protected with warm blankets for transfer to and from the operating room and undue chilling meticulously avoided. Fear may be assuaged by the sympathetic attitude of the surgeon and nurse and by proper sedation. There is no excuse for a child to be brought into the anesthesia room struggling and hysterical. The choice of anesthetic is discussed in the chapter on Anesthesia.

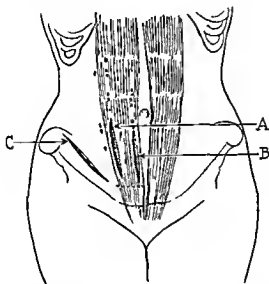


FIG. 173. Incisions commonly employed for appendectomy: *A*, Right paramedian; *B*, right pararectal; *C*, McBurney's incision. The dotted lines indicate the directions in which the incisions may be enlarged.

**Abdominal Preparation** This is unnecessary until the patient is anesthetized. There are numerous skin antiseptics of variable virtue. At the New York Post Graduate Hospital fresh half strength tincture of iodine is employed as follows: a few drops are placed on the umbilicus and the abdomen is thoroughly painted; the entire area is then sponged with alcohol. Care is taken to thoroughly remove all traces of iodine especially in the groins for the child's skin is readily blistered. The abdomen is then dried and ready for operation.

**Choice of Incision** Various incisions are favored by different operators (Fig. 173). The *McBurney* or *griliron* incision with separation of the external oblique aponeurosis and the internal oblique and transversalis muscles in the respective direction of their fibers is an excellent technique. However it is ill suited to cases



requiring intraperitoneal drainage, and sloughing of the aponeurosis with resultant hernia formation occurs more often than with a split rectus incision. The incision is also insufficient for general abdominal exploration. The paramedian incision is excellent for interval appendectomy.

The right pararectus incision is preferable for the following reasons: approach is rapid and relatively bloodless, ample exposure for

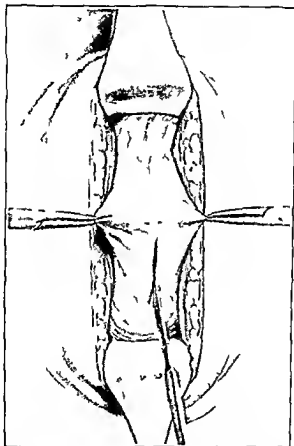


FIG. 174 — The peritoneal cavity is entered by an opening made in the wall over the fascia and peritoneum on the sloping surface at a slight distance from the artery; forceps in order to avoid injury to the gut.

exploration may be obtained by enlarging the incision in either or both directions, closure is simple and rapid, firm healing occurs, drainage is readily secured, pocketing of pus in the mural spaces is uncommon and sloughing of the rectus sheath with resulting ventral hernia is definitely less frequent.

After the incision has been deepened to the rectus sheath, the skin edges are protected by saline pads held with towel clamps.

The rectus sheath and fibers are then divided in the vertical axis and gently retracted. To avoid injury to the bowel, the posterior rectus sheath and peritoneum are elevated by two mouse-tooth

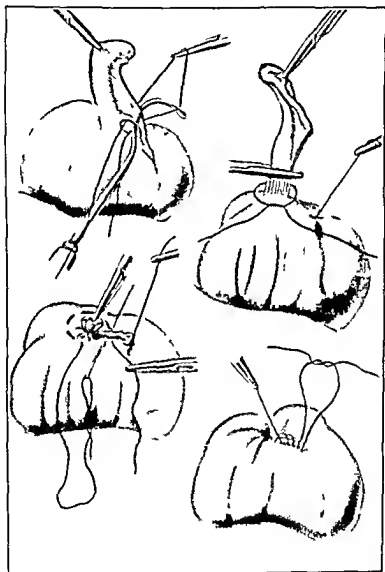


FIG. 175.—Technic of open appendectomy employed when possible

forceps and opened through the sloping surface distant from the forceps, during inspiration (Fig. 174). The peritoneal edges are then clamped with artery forceps and the opening is enlarged with the scissors or scalpel. Blunt tearing and forceful separation of

tissues should be avoided. Retractors are gently insinuated and traction kindly sustained.

**Location of the Appendix** — In clean cases the appendix may often be seen. If not, the index finger is inserted to the pelvic brim and carried from within outward under the cecum thus contracting the meso-appendix and bringing the organ upward or the anterior tunic may be traced downward to the appendix base. At times it is necessary to elevate the cecum into the wound and then locate the appendix which may be retrocecal or retrocolic.

**Technic of Appendectomy** — After the appendix and its mesentery have been thoroughly freed of adhesions the meso-appendix is

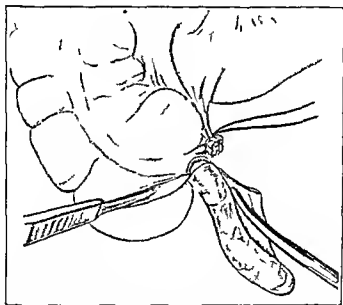


FIG. 176 — Subserous appendectomy. A valuable method in acute appendicitis when the organ cannot be readily delivered. The mesentericum is first ligated and divided. The serosa is then incised circumferentially.

ligated and divided. (The vessels run in the posterior layer of the mesentery.) The base of the appendix is then crushed and ligated and while the organ is held vertically, a purse string is passed of No. 0 or No. 1 plain catgut fused on its needle. An artery clamp is next applied to the appendix distal to the basilar ligature and the organ is amputated with a carbolyzed scalpel. The stump is cauterized with 95 per cent phenol neutralized with alcohol and buried within the purse string. (Fig. 175.) With this technic the former appendix site is completely peritonealized and there are no remaining raw surfaces to promote subsequent adhesions. The ligated and buried stump is also a double safeguard against leakage. Cases in

which the organ cannot be readily delivered may be treated by subserous appendectomy (Figs 176 to 178)

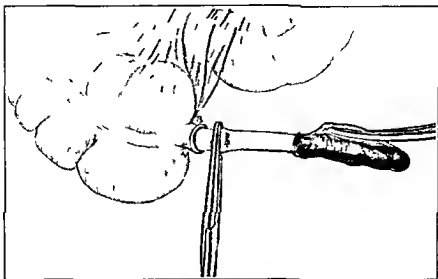


FIG 177 —The serosa with its mesentery being peeled back from the inner tube of muscularis and mucosa. The base is crushed preliminary to ligation.

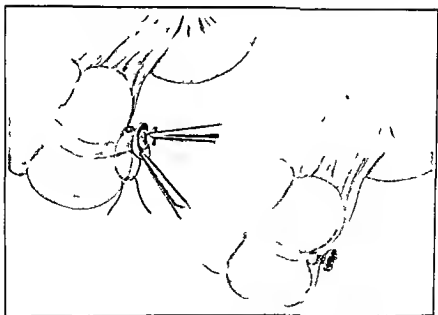


FIG 178 —The ligated stump is embedded within the cuff of serosa, the ends of the mesenteric ligature being employed to close the serosa.

**Closure of the Wound** — After the cecal site has been reviewed for oozing the omentum is drawn gently over it. The peritoneum and posterior rectus sheath are meticulously closed with a continuous suture of No. 1 plain catgut, the muscle edges loosely approximated with two or more interrupted No. 1 plain catgut stitches, the anterior sheath with No. 1 chromic continuous suture, and the skin with fine dermal. In young children retention sutures of non absorbable material are also advisable. The skin sutures are removed on the seventh or eighth day and the retention on the tenth or twelfth.

**When a Small Mass is Palpable** — In such cases the intraperitoneal approach must be guarded for it is impossible to foretell whether the mass consists of edematous omentum and intestinal coils matted about the appendix or an actual abscess is present. The parts extraneous to the inflammatory zone should therefore be carefully protected by saline pads. Free turbid exudate is undisturbed as it is generally sterile and definitely protective. The mass is then explored by gently separating the overlying omentum. If frank pus is encountered it should be immediately aspirated or sponged away and a culture taken. The appendix is then liberated and removed in the manner previously described. Occasionally the cecal wall is too edematous to permit of purse-string inversion of the appendix stump in which event simple ligation must suffice.

Under no conditions should the surrounding fibroplastic adhesions be separated beyond the necessity of liberating the appendix. If frank pus is not encountered intraperitoneal drainage is contra-indicated, only abscess cavities and the presence of excessive necrotic material require drainage and in such cases a single cigarette drain suffices. The pelvis need not be explored unless the preoperative rectal examination has revealed acute tenderness or a mass. The peritoneum and soft parts are closed in layers about the drain. Two or more retention sutures of non absorbable material are usually employed. The writer uses Davey buttons for their anchorage.

**In the Presence of Early Peritonitis** — The purulent exudate which often wells into the wound when the peritoneum is opened is removed by aspiration. The appendix is then removed in the usual manner. After completion of the peritoneal toilet the pelvis should always be explored with the sucker as a pelvic accumulation of pus may lead to secondary abscess formation. Cigarette drainage of the pelvis is advisable in such cases and a second drain is placed in the cecal fossa.

**In the Presence of Diffuse Peritonitis** — It is impossible to drain diffuse peritonitis and in such cases the peritoneal cavity is preferably closed without drainage providing the appendix has been removed and there is no excessive amount of necrotic tissue. Mural drainage is required however and a rubber drain or small cigarette

drain should be inserted to the peritoneum the rectus muscle sheath and skin are approximated about it. Intraperitoneal drainage is definitely indicated when the appendix is left *in situ* and also in the presence of abundant necrotic material. A cigarette drain to the cecal fossa and a second one to the pelvis suffice. The subject of drainage is discussed in the section on Peritonitis.

**In Desperately Sick Patients**—When the appendix is not readily removable, simple incision and drainage is preferable to a prolonged operation with appendectomy. Murphy's dictum to "get in quick and get out quicker" is especially applicable to children. If necessary the appendix may be removed at a later optimum period.

**Cases of Acute Ascending Infection.**—The operative mortality of peritonitis cases of over forty-eight hours duration with acute ascending infection exceeds 70 per cent. These patients exhibit rapid pulse and respiration, mental torpor or delirium and especially slight cyanosis. Predicated upon the principle of absolute peristaltic rest in the Fowler posture, the Ochsner treatment has greatly reduced the death rate of such cases. The advantages of this therapy in certain types of peritonitis appear to be insufficiently appreciated. (The therapeutic details are discussed in the section on Peritonitis.)

**Postoperative Treatment.** The anesthesiologist remains in attendance until the patient is returned to bed. The latter should be warmed and the room free from drafts or cold air. Constant bedside nursing is imperative until the patient has fully recovered from the anesthetic.

**1. Clean Cases**—The patients are placed flat in bed and preferably turned a little on the right side. Adequate aeration is extremely important. A proctoclysis of 2 to 3 ounces of warm tap water with 10 per cent glucose may be given while the anesthetic is wearing off. If the patient is not vomiting, drain doses of warm water may be allowed every thirty minutes the first twenty-four hours. For troublesome vomiting, an antolavage of 3 to 6 ounces of 1 per cent bicarbonate of soda solution often suffices. Excessive vomiting is best treated by siphonage through an indwelling intranasal Levine tube.

**Sedation.**—Operates are unnecessary except for severe postoperative pain. Codeine or small doses of pantopon administered hypodermically usually suffices in older children; appropriate doses of morphine and atropine may be required. The barbiturates are excellent for restlessness.

**Maintenance of the Normal Fluid Balance**—This is imperatively essential. When necessary the oral intake may be supplemented by proctoclyses of tap water with 5 per cent glucose or hypodermoclyses or phleboclyses of 3 per cent glucose in physiologic saline solution. (Proctoclyses are messy and unsatisfactory in young children.)

On the second day the patient is generally quite comfortable. Water, soda pop, albumin water and fruit juice drinks may be given in small amounts. Iced drinks produce cramps. Gas pains may be relieved by the rectal tube and if necessary a warm soap water enema may be given. On the third day gruels, broths, malted milk and junket may be added and on the fourth milk, toast, custard and ice cream. By the seventh day full diet is resumed.

Laxatives are unnecessary and often cause cramps. A daily enema of warm soap water is preferable given by the gravity method with funnel and tube. Distention is best controlled by enemata, hot stupes and colon irrigations. Milk and molasses is a very effective type of enema. Surgical pituitrin or eserine is rarely required.

Children should be allowed to move about in bed. Nature is a ideal guide. In the presence of pain they remain quiet and as healing progresses activity is resumed. Older children are allowed to sit in bed on the fifth day, in a chair on the seventh and to walk on the eighth. Young patients with thin abdominal walls are kept in bed for two weeks.

**B Cases Complicated by Peritonitis.** The postoperative treatment of the e cases is described in Chapter XXXI.

## REGIONAL ILEITIS

### (CROHN'S DISEASE)

Regional ileitis is a definite clinical and pathologic entity of unknown origin. Young male adults are chiefly affected.

The granulomatous process generally involves the terminal ileum and at times spreads to the cecum and portion of the ascending colon. Ulcerotic changes occur in the ulcerative process and great narrowing of the ileum results. The regional lymph nodes may become involved and there is a tendency to fistulous formation between neighboring intestinal coils and occasionally the abdominal wall.

There is usually a history of recurrent attacks of colicky, periumbilical pain, tenderness in the right lower quadrant and intermittent fever. Diarrhea is common. The stools commonly show occult blood and frank melenæ may occur. The sausage-shaped mass in the cecal region may suggest appendicitis, tuberculosis or actinomycosis. Roentgenograms exhibit a string like constriction of the ileum, most often the terminal segment.

Palliative measures are usually futile. Resection of the diseased ileum including the cecum and lower ascending colon when involved is curative. Shortcircuiting of the process may be indicated as a temporary measure in cases with sinuses.

## CHAPTER XXXI

### PERITONITIS

**General Considerations** — The surface area of the peritoneum approximates that of the skin and its cells are capable of absorbing 3 to 8 per cent of the body weight in one hour. This absorptivity potential varies with different portions of the peritoneal cavity, being greatest from the diaphragm progressively less from the omentum, visceral peritoneum and parietal peritoneum, and least from the pelvis. The Fowler posture minimizes absorption by approximately 10 per cent.

**Factors Influencing Absorption** — The rate of absorption is influenced by several factors: vascularity of the peritoneum, the character of the fluid, its localization, effect of gravity, and especially peristaltic activity. It is definitely decreased by the cessation of peristalsis. The absorption of soluble toxins parallels that of fluids and operative procedures in ascending peritoneal infections may precipitate a lethal dose of toxin and cause death within a few hours. Such mortalities are often attributed to shock.

**Resistance of the Peritoneum to Infection** — This is definitely reduced in infancy, also in asthenia, nephritis, diabetes, obesity, dehydration, and in the presence of blood clots. Normally the bowel wall is impervious to the transmission of bacteria but following trauma or circulatory interference, peritonitis may develop without a break in surface continuity. While the dominant cause of peritoneal invasion is perforative appendicitis (98 per cent), it may also result from tuberculosis or typhoid ulceration, strangulation of the gut in hernia, volvulus, or intussusception, or ulceration of Meckel's diverticulum.

**Peritoneal Response to Bacterial Invasion** — This response is evidenced by an immediate and active transudation followed by exudation. The fluid at first clear, becomes progressively turbid and purulent. The fibroplastic elements in the exudate cause adherence of adjacent intestinal surfaces and aided by the omentum, tend to wall off and delimit the septic focus. This glialytic cohesion occurs very rapidly, at times within one-half hour. Organization of the exudate at a later date may result in adhesion formation. Necrotic material is removed by the phagocytic endothelial cells of the peritoneum, macrophages, microphages and the neutrophils. Foreign bodies are usually encapsulated by fibroplastic lymph.



**Defense Processes**—Peritoneal transudate plastic exudate and fresh pus are bacterioidal bacteriolytic and antitoxic. The exudate on the outskirts of the infected zone is often sterile. Whereas this is essentially protective and should not be sponged away or aspirated the focal contaminated pus containing dead cells requires evacuation. It should be emphasized that the protective exudative processes of the peritoneum are definitely impaired by handling sponging exposure to the air and washing with saline or antiseptic solutions. Delimiting adhesions are defensive processes and should not be separated.

**Peritoneal Response to Different Organisms** Peritoneal insult at first produces hyperperistalsis. This is later followed by an arrest of peristaltic action (adynamic ileus) with concomitant distention and failure to pass flatus or feces. The peritoneal response to different organisms is varied and specific. *B. coli* produces abundant dirty yellow pus with a characteristic foul odor. In pure culture it is highly virulent and localizing adhesions are wanting. The organism however is usually associated with the staphylococci and the latter produces abundant delimiting adhesions which tend to localize the peritonitis and moderate its severity. The statistical frequency of colon group infections may not represent actual etiologic conditions. In many instances the *B. coli* is a secondary invader the primary causative agent being the streptococcus. By outgrowing the initial organism the *B. coli* becomes pre dominant. (Refer to Bacteriology under Appendicitis.)

*Streptococcus hemolyticus* varies greatly in virulence and becomes especially lethal during influenza epidemics. It produces a serous exudate at times sanguinous and without odor. Creamy pus may be associated with less virulent strains. Non hemolytic streptococcus likewise varies greatly in virulence. Severe infections produce little or no exudate give a blistered appearance to the peritoneum and produce large quantities of toxin. In addition to surface extension the infection spreads through the subserous lymphatics with resulting subperitoneal cellulitis and not infrequently septicemia. Streptococcus infections following perforative appendicitis are generally of only moderate severity.

*Staphylococcus albus* is at times non pathogenic. It is generally of slight virulence and produces a milky odorless pus with excessive fibroplastic elements. Commonly associated with *B. coli* in appendicitis it occupies an indeterminate position in the production of peritonitis. The organisms occur chiefly on the outskirts of the inflammatory zone and assume a protective role by creating delimiting adhesions. The *Staphylococcus aureus* is somewhat more virulent.

*Pneumococcus* produces a thick odorless yellow green gummy pus which contains abundant fibrin flakes and masses. The infection may be mild and localized or extremely virulent and diffuse.

*B. pyocyaneus* a normal habitat of the intestines generally induces a local peritonitis. Green pus is only produced in the presence of oxygen which is essential for the production of the pigment pyocyanin. The organism is never associated with the colon bacillus.

*B. aerogenes encapsulatus* never produces peritonitis but may complicate wound infections.

The pathogenicity of the intestinal flora increases in virulence from the duodenum downward and the most virulent types of bacteria are found in the terminal ileum, cecum and ascending colon. The colon bacillus (associated with the staphylococcus) is the dominant statistical organism in peritonitis and the streptococcus is the next most frequent.

**Classification of Peritonitis**—The varieties of peritonitis may be classified according to

(a) *Duration*—Acute, subacute and chronic (tuberculosis, actinomycosis).

(b) *Situation*—Local or circumscribed (abscess), diffuse or spreading, general and pelvic.

(c) *Etiology*—Pyogenic (*B. coli*, streptococcus, staphylococcus, pneumococcus, gonococcus, pyocyaneus), hematogenic (tuberculous, actinomycotic), enteric (*B. typhosus* and anaerobes) and radiogenic.

(d) *Primary*—Due to undiscoverable nidus (pneumococcus).

**Acute Suppurative Peritonitis** — This type is characterized by the presence of a protective exudate which forms delimiting adhesions and the roughened and hyperemic peritoneum is covered with fibroplastic material. The condition occurs most commonly with combined *B. coli* and *staphylococcus* infections following perforative appendicitis less frequently with *streptococcus* and infrequently with *pneumococcus* invasion.

**Symptomatology of Acute Peritonitis** — Peritonitis is almost always a complication of some other intra abdominal pathology and occurs as a secondary or terminal event. *The cardinal symptoms are pain, tenderness, abdominal rigidity and muscle spasm.* The pain is constant in character, tends to be localized in the early stages and is increased by movement or pressure. The tenderness which at first may be general soon becomes localized over the inflammatory zone and is accompanied by definite focalized abdominal rigidity and muscle spasm. In young children rigidity is an uncertain factor and focal hypersensitiveness of the skin is often more readily demonstrable. Rebound tenderness is a highly valuable diagnostic sign. The area of tenderness and protective muscle spasm increases in accordance with the spread of infection.

*Emutting* often occurs at the onset and may be aggravated by taking water or medicine. The vomitus consists of stomach contents and bile. In the later stages (adynamic ileus) it may contain upper intestinal contents and occurs without effort. *Fever* is usually present; it is seldom high and its degree is no index of the pathologic severity. The *pulse rate* rises rapidly and the *respirations* become shallow due to splinting of the abdominal muscles.

**Blood Examination** — *Leukocytosis* and *polynucleosis* accompany the process except in fulminant *streptococcus* infections. The average white blood cell count varies from 15,000 to 25,000 and the polymorphonuclears from 75 to 90 per cent. (The normal blood count variations occurring in infants and young children are discussed in the section on Appendicitis.) Bacteriemia is rarely present except in terminal sepsis.

The Schilling leukocyte count and sedimentation test are of corroborative value. The normal percentage of immature white cells in children is 10 per cent or less; a count of 10 to 15 per cent suggests an inflammatory process and over 15 per cent a progressive one. During the conservative treatment of peritonitis (between the second and seventh day) the Schilling count may be of considerable value in estimating the progress of the infection. It is also helpful in the unusual types which are afebrile and exhibit only a slight disturbance of pulse rate. The sedimentation test is abnormal in peritonitis and also in acute pulmonary inflammations. A normal reaction (with few exceptions) excludes the presence of peritoneal inflammation.

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(b) *Situation* Local or circumscribed (abscess) diffuse or spreading general and pelvic

(c) *Etiology* —Pyogenic (*B. coli* streptococcus staphylococcus pneumococcus gonococcus pyocyaneus) hematogenic (tuberculous actinomycotic) enteric (*B. typhosus* and anaerobes) and radiogenic

(d) *Primary* Due to undiscoverable nidus (pneumococcus streptococcus)

*Secondary* —(a) Usual type secondary to perforation rupture leakage of bacteria or irritating fluids and (b) secondary to septicemia in pneumonia or sepsis (often terminal)

**Clinical Classification** For clinical purposes acute peritonitis is best classified according to the character of the exudate as follows (a) Acute septic peritonitis and (b) acute suppurative peritonitis. In either type the process may be local or diffuse. General peritonitis rarely occurs as the entire greater and lesser sacs are seldom involved.

**Acute Septic Peritonitis** —This is due to the streptococcus. The process is a diffuse ascending infection which often produces lethal amounts of toxin. The peritoneum exhibits a blistered appearance lusterless edematous and markedly hyperemic even with petechiae. There is little or no protective exudate and delimiting adhesions are wanting. In addition to surface spread the organisms invade the subserous lymph spaces and the subperitoneal cellulitis produced thereby is often associated with septicemia. The crises occur most frequently in children suffering from septic throats erysipelas scarlet fever otitis media and sepsis or as a result of contaminating infection occurring during operation. The vast majority succumb within ninety-six hours and in the writer's opinion operative interference is contraindicated. The Ochsner form of expectant therapy is preferred.

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**Stage of Progressive Toxemia.**—Pathologic processes progress rapidly in early life and the patient soon passes into the stage of progressive toxemia. The facies becomes anxious, pinched and pallid, the eyes bright and sunken with circumoral pallor, and the cheeks flushed. The spreading tenderness and board-like rigidity are accompanied by the development of paralytic ileus and the abdomen becomes distended and tympanitic. The vomiting becomes projectile or of the spitting spill-over effortless type, copious in amount, and either black from extravasated blood or yellowish and offensive from regurgitation of the intestinal contents. Visible and auscultatory peristalsis are absent and enemas are retained or returned without gas. In pneumococcus and streptococcus infections, toxic diarrhea may occur.

**The Terminal Stage**—In the terminal stage the Hippocratic facies develops: anxious pinched expression with sharpened nose, sunken eyes, grayish collapsed cheeks and cold sweating brow. Rigidity may disappear and tenderness lessen. The temperature may remain moderately elevated or rise to  $106^{\circ}$  F., and the pulse becomes extremely rapid and feeble. The sensorium remains clear until death. Blood cultures are often positive, the bacteremia being an evidence of terminal sepsis.

**Diagnosis of Peritonitis.**—The history is of utmost importance: Was the child perfectly well before the attack began, have there been previous attacks of "indigestion," and especially, has a physic been given? (An inflamed appendix seldom ruptures without a cathartic being administered and castor oil is the chief offender.) Was the onset sudden, was pain the first symptom, where did it begin and has it been constant? When and what character was the last bowel movement and was it accompanied by blood or mucus? Has there been any vesical irritability? (This occurs commonly in young children.)

**Inspection**—Much can be gained by observation: A child with peritonitis lies still on its back or side, with thighs flexed and knees drawn up, carefully splinting the abdomen—any movement increases the pain. The respirations are shallow and thoracic in type and unaccompanied by dilatation of the alae nasi or expiratory grunt. The pain is constant and there are no free intervals from it as occur in intussusception. The distention is symmetrical and there is no visible peristalsis. The patient is fearful and the surgeon's approach should be made with care and gentleness.

**Palpation**—The flank muscles should always be carefully examined as well as the front of the abdomen. Tenderness, if carefully elicited, is a reliable sign and usually indicates the site of lesion. Interval examinations are extremely important in determining the spread of tenderness (of the infection). Abdominal rigidity is inconstant in young children and cutaneous hyperesthesia is more often

*demonstrable* Rectal examination if properly performed should not excite the patient the child's pelvis is small and tenderness or the presence of a mass is readily elicited

**Auscultation**—Absence of auscultatory peristalsis is a valuable sign a silent acutely distended and tender abdomen is ominous A thorough chest examination should always be made to exclude pneumonia or pleurisy Intractable and highly neurotic children occasionally require light narcosis for satisfactory abdominal examination

**Temperature and Pulse**—The temperature is variable and unreliable It should be emphasized that in unusual instances diffuse peritonitis may be afebrile The pulse rate and its character are highly important (Average pulse rates for different ages are discussed in Chapter I) In the early stages of peritonitis the pulse is wiry but becomes feeble and thready as the process diffuses *Progressive increasing pulse rate denotes advancing infection*

**Dehydration** occurs early from the vomiting of fluids and the absence of absorption by the inflamed intestines (Refer to Dehydration) The condition of the tongue is a valuable guide the return of moisture to the parched organ is an early sign of subsiding peritonitis especially when associated with a lowered pulse rate

**Differential Diagnosis**—Since acute peritonitis in children is a complication of appendicitis in over 98 per cent of cases the differential diagnosis is discussed in the chapter on Appendicitis

**Prognosis of Acute Peritonitis** The prognosis is poorest in infants and young children Generally speaking cases of diffuse peritonitis operated upon within twelve hours offer a favorable outcome between twelve and forty-eight hours the mortality rises rapidly and after forty eight hours the outlook is grave Pneumococcus and streptococcus infections are often fatal also pure B coli Mixed colon and staphylococcus infections are less serious and the gonococcus is rarely lethal Clinically the order of virulence from the least to the highest is as follows gonococcus perforative appendicitis gut perforation from trauma or strangulation pneumococcus streptococcus and general sepsis

The following are ominous continued rising pulse rate progressive hyperpyrexia persistent distention and obstipation toxic diarrhea stercoraceous vomit continued hiccough jaundice bacteriemia and repeated chills indicating pyelophlebitis

The average mortality for all types of diffuse peritonitis is 5 to 15 per cent Death occurs from (1) toxemia resulting from peritoneal absorption (2) absorption of enterogenic toxins in adynamic or dynamic ileus (3) septicemia and (4) complications—pulmonary hepatic cardiac pyemia subphrenic abscess and dehiscence

**The Treatment of Peritonitis**—The treatment of peritonitis in the early stages is immediate operation and removal of the cause

when possible. The products of purulent inflammation under pressure are extremely toxic and prompt relief by drainage is the essence of surgical intervention. The entire pathologic zone need not be subjected to exposure nor is it always imperative that the causative agent generally the appendix be removed. The essential is to institute peritoneal drainage and Murphy's dictum to "get in quick and get out quicker" is especially applicable to children.

**Exceptions to Immediate Operation.** Operation should be delayed in (1) *Pneumococcus peritonitis* (2) overwhelming *streptococcus peritonitis* following septic throats (3) *peritonitis* secondary to operation or to general sepsis (4) *gonococcus peritonitis* and (5) cases of over forty-eight hours' duration in which there is ascending infection.

The superior statistics of English clinics are largely due to the adoption of conservative supportive treatment during the intermediate inflammatory septic stage (second to seventh day). The writer believes that in this stage of acute ascending infection the Ochsner treatment will salvage more lives than immediate operative interference.

**Ochsner Treatment.**—Its purpose is to produce localization of the peritonitis through absolute peristaltic rest during the dangerous intermediate period of ascending infection. The patient is placed in the Fowler posture with young children the head of the bed is elevated 30 degrees. Nothing not even sips of water is given by mouth and peristaltic rest and amelioration of pain are secured by the administration of codeine, pantopon or small repeated doses of morphine. Adequate water and salt balance is maintained by hypodermoclyses or infusions of normal saline solution with glucose (Refer to Treatment of Dehydration). In ten to fourteen days when the process has localized and the pulse and temperature have become stabilized the pus is evacuated and the abscess cavity drained.

**Operative Treatment.**—Local and spinal anesthesia are impractical in children and inhalation anesthesia skilfully administered in respect to aeration and relaxation is definitely preferable (Refer to Anesthesia). The cause should be removed when possible—in appendicitis appendectomy should be performed unless unusual mechanical difficulties obtain a perforation of the bowel should be closed and gangrenous gut should be resected or treated by the Mikulicz method. Evisceration should be prevented and extensive exploration omitted. *Delimiting adhesions* should be left undisturbed and any unnecessary handling exposure sponging or washing of gut carefully avoided. Contaminated pus and necrotic material including accumulations in the pelvis should be aspirated. Saline solution and antiseptics should not be poured into the peritoneal cavity.



**Drainage**—It is impossible to drain the entire peritoneal cavity and in most cases of diffuse peritonitis the abdomen is best closed without intraperitoneal drainage. Mural drainage is necessary however and a small cigarette or folded rubber dam drain is inserted down to but not into the peritoneum. In the presence of abundant necrotic tissue about the appendiceal area a single drain to the cecal fossa is indicated and in cases with large accumulation of pus in the pelvis a second cigarette drain is inserted into the pelvis. The former usage of multiple intraperitoneal drains availed nothing from the standpoint of actual drainage. They not only often produced mechanical obstruction and at times intestinal fistulæ but also caused the later development of adhesions with varied sequelæ. The omission of unnecessary drainage in diffuse peritonitis has definitely reduced the mortality and lessened complications. It has been repeatedly established that much of the purulent exudate in peritonitis is protective—bactericidal, bacteriolytic and antitoxic.

**Conditions Requiring Drainage**—Drainage is definitely necessary in abscess cavities in the presence of excessive necrotic material or infected blood clots and when the affected appendix is not removed. As much as possible of the pus and necrotic material should be removed by aspiration and gentle sponging (not rubbing) and the cavity drained by one or more soft rubber tubes or cigarette drains; some prefer the Mikulicz perforated rubber dam. The peritoneum is accurately closed about the drain with plain catgut, the fascia and muscles with No. 1 chromic and the skin with dermal or silk. Non absorbable retention sutures should be employed and allowed to remain for two weeks. The gauze dressings are covered firmly with adhesive plaster and reinforced with a muslin binder. Adequate abdominal support is advisable for several days to prevent possible dehiscence. After the drains become loosened on the fifth or sixth day they are shortened about an inch daily. Prolonged drainage may lead to the development of all lesions: sinus formation or fecal fistulæ.

**Duodenal Siphonage**—In the presence of paralytic ileus drainage of the upper intestinal tract is imperative as the absorption of enterogenic toxins (histamine etc.) may prove rapidly lethal. This is so ideally accomplished by duodenal siphon drainage through an indwelling Levine tube that high jejunostomy is seldom resorted to. Costain's lymphaticostomy is valueless and Handley's jejunocecalostomy is too grave a procedure.

**Postoperative Treatment**—The Fowler posture is maintained for several days. In the case of infants the head of the bed is elevated on shock blocks to an angle of 30 degrees. The absorption of toxins is thereby reduced approximately 15 per cent through gravitation of the exudate into the lower abdomen and pelvis.

Nothing including sips of water or cracked ice is given by mouth until the pulse becomes stabilized and gas is passed per rectum (Even small amounts of water stimulate peristalsis) The saline-water balance is adequately maintained by hypodermoclyses or infusions of physiologic salt solution (Some prefer Ringer's or Hartmann's solution) Three to 5 per cent glucose is generally added to the saline solution It is a readily oxidizable food and specifically prevents and combats acidosis (Refer to Dehydration)

Supplementary proctoclyses of 10 per cent glucose in tap water may be given to older children in amounts of 2 to 4 ounces every four hours Nutrient enemata are inadvisable (The administration of excessive amounts of saline solution will produce hydemia and produce edema of the ankles face and lungs)

**Sedation** No opiates are administered except for pain Codeine or small doses of pantopon usually suffice in older children appropriate doses of morphine may be required The barbiturates are excellent for restlessness

**Postoperative Distention** This is best controlled by hot stupes colon irrigations and enemata Milk and molasses is the most effective type of enema Cathartics pituitrin and eserine are distinctly contraindicated in the early stages of peritonitis

**Localization of the Peritonitis**—The localization and subsidence of the peritonitis is evidenced by stabilization of the pulse and temperature cessation of vomiting moistening of the tongue limitation of tenderness and rigidity softening of the abdomen decrease of tympany and especially by the passage of flatus with or without feces This may be tested by giving a hot soap-water enema If the passage of gas and feces is not followed by either abdominal pain or rise of temperature it is safe to give water tea or soda water in dram doses every fifteen minutes

**Resumption of Nourishment**—For the first forty-eight hours after the peritonitis has subsided tap water tea and albumen barley or soda water may be administered in doses up to 2 ounces each hour Ice or iced drinks often produce cramps Gruels broths malted milk junket jello calves foot jelly and custard are gradually added After the fourth day ice cream is allowed and by the tenth day the normal dietary is resumed *Protracted cases with secondary anemia are greatly benefited by blood transfusion*

Laxatives are not necessary A daily enema is preferable either of soap water or equal parts of milk and molasses (Enemas should be given warm and by the gravity method with funnel and tube) In the case of nurslings the mother should have breast pumping at regular intervals to maintain lactation The aid of the pediatrician is invaluable in the postoperative feeding of young patients

## ACUTE LOCAL PERITONITIS

Acute local peritonitis may be the localized residuum of a diffuse process more often however it represents a delimiting defense process about the primary infection its focal character being an evidence of satisfactory response

**Pathology** The pathology resembles that of diffuse peritonitis except for being circumscribed by the delimiting plastic exudate which covers the adjacent peritoneum omentum and intestinal coils. The turbid fluid beyond the zone of localization is generally sterile and definitely protective.

**Symptomatology**—The constitutional reaction is less severe than in diffuse peritonitis. Although the pulse rate and temperature may be considerably elevated they are stabilized and the degree of toxemia is definitely less. Unless the process be in the upper abdomen respiratory excursion is not embarrassed. The local signs are those of focal pain tenderness and rigidity with or without a palpable mass.

Whereas resolution may take place in some instances the process usually eventuates in abscess formation. In the latter event rupture may occur either into a hollow viscus (occasionally followed by spontaneous cure) or into the free peritoneal cavity with resultant diffuse peritonitis. Resistance in the specific organism is generally so well developed that soiling of the peritoneum at operation does not produce a spread of infection.

**Treatment**—This comprises removal of the cause which in over 98 per cent of cases is the appendix and drainage. The subject is discussed under Appendiceal Abscess.

## SECONDARY ABSCESS

Secondary abscess may be the sequel of either local or diffuse peritonitis. The location of the pus is governed largely by gravitational seepage from the primary focus to certain dependent abdominal fossae viz the pelvis right and left lumbar gutters and right and left subphrenic spaces. Due to ineffective drainage pelvic abscess develops more frequently from appendicitis than secondary abscess in the right lumbar gutter. It is rare to find an abscess between coils of intestine.

**Pelvic Abscess**—Considering the frequency of primary pelvic peritonitis in perforative appendicitis the incidence of secondary abscess is comparatively low. Confined to the recto-vesical and recto-uterine pouches in the male and female respectively the upper boundaries of the abscess are formed by intestinal loops and the omentum. The organisms commonly the colon bacillus and staphylococcus are seldom of virulent type and the process pursues a subacute course. Occasionally an abscess resolves, more often

rupture occurs into the abdominal cavity resulting in diffuse peritonitis or into the rectum eventuating in cure. A loop of terminal ileum adherent to the pelvic wall may occasionally cause intestinal obstruction.

**Symptomatology** Secondary abscess in the pelvis is usually well borne. Generally between the seventh and tenth day following operation the temperature rises, moderate distention recurs and the patient complains of abdominal cramps indefinitely localized. Increasing vesical irritability is a common symptom and enemas become painful. A tender mass is usually palpable upon rectal examination.

Secondary abscess in the right lumbar gutter exhibits focal tenderness and moderate muscle spasm often most pronounced in the flank. The constitutional reaction is seldom marked. Leukocytosis and polymorphonuclear leukocytosis recur.

**Treatment**—Conservative treatment should be employed unless an abscess is definitely demonstrable. Children repeatedly develop symptoms suggestive of secondary abscess and recover spontaneously. *Except for definite pathologies secondary operations are dangerously meddling.*

When fluctuation is palpable in the pelvis the following procedure is employed. The sphincter after having been moderately dilated a blunt artery forceps is insinuated through the rectal wall into the abscess cavity. As soon as pus is obtained its blades are opened and the exudate is evacuated. Insertion of a soft rubber tube into the cavity is advisable for forty-eight hours. Irrigation however is contraindicated. Vaginal drainage through the posterior cul-de-sac is not recommended in young females. Most cases recover rapidly.

An abscess in the right lumbar gutter may be evacuated through either the primary appendectomy incision or a secondary flank approach.

### SUBPHRENIC ABSCESS

Subphrenic abscess may be regarded as a localized secondary peritonitis in which the purulent collection occurs between the diaphragm above and the transverse colon and mesocolon below. The abscess may be supra or infrahepatic. The condition seldom occurs in children and the dominant etiologic factor is perforative appendicitis. It may also be a sequel of pneumonia, empyema, infections of the liver, spleen or kidney, Pott's disease, echinococcus cyst or sepsis. *B. coli* or staphylococci are the usual organisms.

**Pathology** The suprahepatic space is divided into four parts by the falciform, coronary and lateral ligaments. The right posterior superior space limited by the falciform ligament on the left and the coronary and right lateral ligaments anteriorly is involved most

commonly and the infection generally occurs by direct extension from an acutely inflamed high retrocolic appendix (Refer to Appendicitis). The right anterior-superior the extraperitoneal and the left superior spaces are rarely affected. Abscess of the right or left infrahepatic space is generally a sequel of perforation of the gall bladder stomach or duodenum. Such pathologies are rare in early life.

As the suprahepatic abscess increases in size the diaphragm becomes elevated and fixed. If unrelieved the pus may rupture into the pleura lung bronchus pericardium stomach colon or through the parietes. Pleural effusion or frank empyema is a common complication. Not infrequently a gas bubble develops (*B. coli*).

**Symptomatology** —The usual clinical picture is that of the cerebral disease followed in a week or ten days by the development of low grade sepsis. Although at times an initial chill occurs the onset is usually insidious. Gradually there develops continuous remittent fever of  $102^{\circ}$  to  $104^{\circ}$  F. increasing pulse rate sweating and leukocytosis. For several weeks there may be an absence of focal signs and symptoms. Cough produced by pleural exudate may be the first symptom or local soreness or discomfort may finally develop. Occasionally shoulder pain is complained of a reflex phenomenon due to diaphragmatic irritation.

**Physical Signs** —These generally simulate those of pleural effusion (this is often present). Shifting dullness and tympany are only demonstrable in large abscesses which contain considerable gas. Widening of the right costal angle is seldom present. Exploratory puncture through the diaphragm is a dangerous procedure.

**Roentgenography** —*The roentgen ray is the most valuable diagnostic aid.* In the absence of pleural exudate the high immobile and abnormally convex diaphragm is clearly outlined. When gas is present (15 per cent of cases) a crescent bubble above the fluid level may also be exhibited (pathognomonic finding). In the presence of thick pleural exudate however the diaphragm cannot be visualized in the dense shadow. In such cases a roentgenogram following pneumoperitoneum may reveal obliteration of the usual space between the liver and diaphragm. The heart may be elevated without lateral displacement.

**Prognosis** —Following rest and local heat resolution may occur in rare instances. The high operative mortality approximating 20 per cent is largely the result of late diagnosis. Many cases are unrecognized for months. The writer was recently consulted in a case of bronchial fistula which developed sixteen weeks after laparotomy for a perforated retrocolic appendix. Lipiodol injected into the abdominal sinus produced violent paroxysms of coughing and was expectorated in the purulent mucus. A sinus tract was clearly

demonstrable passing through the diaphragm and communicating with a main lower right bronchus. The diagnosis of subphrenic abscess has not been considered.

**Treatment** This comprises surgical evacuation of the pus and adequate drainage of the abscess cavity. When the right posterior superior space is involved a posterior extraperitoneal approach is preferable. Following subperiosteal resection of the twelfth rib a transverse incision is made through the attachment of the diaphragm at the level of the transverse process of the first lumbar vertebra thus entering the extraperitoneal space. The peritoneum is then carefully separated from the diaphragm until the abscess is entered. After evacuation of the pus one or two soft rubber tubes are inserted. Satisfactory drainage usually eventuates in recovery.

If a transpleural approach is elected preliminary fixation of the diaphragm to the costal pleura should be secured. This may be accomplished by a two-stage operation in which after rib resection the wound is packed with gauze for forty-eight hours to promote adhesions between the diaphragm and chest wall. Such adhesions are undependable however and it is safer to secure the diaphragm to the chest wall by sutures. At the second operation the diaphragm is incised and the abscess cavity evacuated and drained.

Neither the retroperitoneal nor transpleural approach is attended by untoward shock if skilfully performed. Early diagnosis and operation greatly reduce the mortality.

### STREPTOCOCCUS PERITONITIS

The streptococcus usually occurs in conjunction with other organisms (symbiosis) and the severity of infection is modified thereby. As a single invader in postoperative peritonitis and in cases following septic throats erysipelas scarlet fever otitis media influenza empyema or sepsis it may produce the most virulent type of peritonitis.

**Pathology** — Acute diffuse peritonitis occurs more often than with any other organism and massive amounts of lethal toxins are rapidly produced. The peritoneum appears blistered red injected edematous and lustreless there is but little turbid or at times serosanguinous exudate and fibroplastic elements and delimiting adhesions are absent. In addition to surface spread the organisms permeate the peritoneum and invade the subserous spaces. The resulting subperitoneal cellulitis is frequently accompanied by septicemia.

**Symptomatology** — The constitutional reaction to the grave toxemia far outweighs the focal abdominal signs. At the onset there may be a chill the severe pain and vomiting are followed by hyperpyrexia rapid progressively increasing pulse rate and prostration.

Diffuse tenderness and rigidity develop early. In fulminating cases the patient is overwhelmed and death occurs in forty eight to seventy two hours. Terminal blood cultures are generally positive. In exceptional instances of less virulent type the peritonitis may become localized.

**Treatment**—Operation in fulminant streptococcus peritonitis is futile. The Ochsner method of treatment with vigorous supportive measures including blood transfusion probably offers the best prospect of recovery.

### POSTOPERATIVE SEPTIC PERITONITIS

This tragic pathology may result from various causes. (1) As a sequel of acute intra abdominal inflammation *i. e.* following appendectomy for acute appendicitis. (2) faulty operating room technique. (3) contaminated catgut and (4) infection carried by a member of the operating team. The latter may result from insufficient scrubbing touching an unsterile object talking or coughing into the wound or inadequate masking of the nose and mouth.

**Pathology**—The peritonitis is almost always due to streptococcus infection. The peritoneum appears blistered lusterless edematous and injected often with petechiae the exudate is thin odorless scant in amount and at times blood tinged and there is little or no fibrin formation or delimiting adhesions. The process is a fulminating diffuse infection in addition to surface spread the organisms invade the subserous lymph spaces and the resulting subperitoneal cellulitis commonly eventuates in septicemia. Lethal amounts of toxin are produced rapidly and death results generally within ninety six hours.

**Symptomatology**—The classical features of peritonitis are absent or so slight that they may escape recognition. There may be no special untoward symptoms or focal signs during the first forty eight hours after operation the pain temperature tenderness and distention are often no more pronounced than occur in severe normal postoperative reactions. The cardinal symptoms are progressive toxemia and rapid rising pulse rate. Obstipation becomes complete and enemas are either retained or returned without flatus or feces. Vomiting due to paralytic ileus may be a late symptom. Death usually occurs on the third or fourth day and many such mortalities are erroneously attributed to postoperative pneumonia.

**Diagnosis**—In addition to the progressive toxemia and rising pulse rate careful abdominal examination will generally reveal greater tenderness and muscle spasm in the flanks than over the anterior abdomen the latter often appears more doughy than rigid. Any movement of the patient increases the pain. In contradistinction to mechanical intestinal obstruction there is an absence of

both visible and auscultatory peristalsis. Terminal bacteremia is usually demonstrable.

**Prognosis** — Practically all cases succumb.

**Treatment** — This comprises supportive measures. (Refer to Treatment of Peritonitis.) Operative interference is futile except when there is leakage from a hollow viscus.

### PERITONITIS IN THE NEW-BORN

The condition may occur at birth or develop during the first weeks of life as a complication of neonatal sepsis. The exciting organism is generally the streptococcus, less often the staphylococcus and rarely the gonococcus or pneumococcus. The portal of entry is commonly the umbilicus although it may be any wound in the skin or mucous membranes. Contaminated milk from mothers suffering from mastitis or septicemia is rarely a factor.

**Pathology** — In most instances the peritonitis is diffuse and resembles that of streptococcus infections in older children. At times, however, the process is localized in the region of the umbilicus and an abscess forms.

**Symptomatology** — The onset may be insidious with vomiting, progressive distention, dilatation of the abdominal veins and gradual development of a mass in the periumbilical region. Generally, however, the clinical course is more acute and definite, persistent vomiting, rapid distention, abdominal rigidity and flexion of the thighs. Bacteremia is not uncommon. Cases with diffuse peritonitis succumb rapidly.

**Treatment** — Except in the mild types of focal abscess which require incision and drainage, the treatment is entirely supportive through the administration of saline-glucose clises and repeated blood transfusions.

### TUBERCULOUS PERITONITIS

Tuberculous peritonitis is the most common type of chronic peritonitis. It is predominantly a disease of children and young adults and the majority of cases occur between the second and sixth years. The pathology seldom develops in sucklings or in infants under one year except as a concomitant lesion of acute miliary tuberculosis. Many mild infections apparently become healed without producing symptoms of recognition, for at necropsy approximately 18 per cent of tuberculous children exhibit occasional peritoneal tubercles (Bildert). Congenital cases have been observed. The clinical incidence of the disease varies from 0.1 to 0.25 per cent of sick children.

**Modes of Infection** — (1) *Alimentary tract* (a) In open pulmonary lesions sputum may be swallowed. (b) About 30 per cent of



infections are due to the ingestion of bovine bacilli in contaminated milk and milk products. Primary localization may occur in the intestine, mesenteric lymph nodes or appendix. (2) *Blood and lymphatics*. A concomitant of general military tuberculosis. (3) *Genital tract*. From the Fallopian tubes in tuberculous salpingitis. Leakage of bacteria into the peritoneal cavity may continue for a long time as the fimbriae do not become occluded as in gonorrheal salpingitis. Although the peritonitis often appears as the sole clinical manifestation of tuberculosis, the pathology is always secondary to some primary nidus.

**Pathology**—Gray military tubercles varying in size from minute specks to 1 cm. in diameter are disseminated diffusely over the surface of the peritoneum. Their invasion of the peritoneum produces fibrinous exudate in a varying amount of serous, seropurulent or hemorrhagic effusion. The relative amounts of fibrin and fluid vary in different types. In the ascitic form, fluid predominates and adhesions are minimum and easily separated, in the fibrinous form fluid is scant and intestinal loops become firmly adherent to each other and to neighboring viscera. Most cases exhibit both conditions in varying degree. The tubercles may coalesce and caseate, or heal and completely disappear.

The omentum may be thickened and contain large nodules or be shrunk and firmly rolled about the transverse colon. An extensive adenopathy of the mesenteric nodes may also occur (tabes mesenterica). In any of the foregoing types, ascitic, fibrinous or tabes mesenterica, large caseous masses may develop and through encapsulation produce cold abscesses. The latter may ulcerate into the intestine or discharge through the abdominal wall commonly about the navel. At any stage of the pathology intestinal obstruction may develop.

**Classification**—Pathologic varieties, based upon the dominant lesion, are usually described as follows: (1) Ascitic or exudative, (2) fibrinous, adhesive or plastic, (3) tabes mesenterica, and (4) ulcerative or caseous. There is no clear-cut individual pathologic type, however, and most cases exhibit a combination of (1) and (2). The ascitic form predominates in about 70 per cent of cases, the fibrinous in 25 per cent and the ulcerative in 5 per cent. The clinical classification of acute and chronic peritonitis is seldom employed. Cases of the former are of rare occurrence except in acute military tuberculosis, and the peritoneal response is minimum.

**Symptomatology**—Although no sharp distinction can be made between the various types each pathologic form will be discussed separately for purposes of emphasis and clarity.

1. **The Ascitic Form**—Gradual increasing distention of the abdomen may be the first symptom to attract attention. Often there is prodromatory malaise, anorexia, loss of weight, irregular fever,

indefinite digestive disturbances and attacks of diarrhea. At first the abdomen is tympanitic. Later evidences of free fluid are exhibited—shifting dullness in the flanks, upward displacement of the diaphragm and of hepatic dullness, pointing umbilicus and dilatation of the abdominal veins. The aspirated fluid is usually straw-colored but may be slightly brownish or blood tinged. Its specific gravity varies from 1.018 to 1.026, the fibrin content is low, and the cellular elements are chiefly lymphocytes. Guinea pigs generally develop tuberculosis if the fluid is injected intraperitoneally.

The omentum is often thickened and palpable as a broad transverse roll across the upper abdomen, and enlarged mesenteric glands may form irregular masses. Although pigmentation may develop and become quite general, the buccal mucous membrane is never involved. The constitutional reaction is slight.

Invasion of the peritoneal cavity by the tubercle bacillus may at times produce acute symptoms—pain, nausea, vomiting, focal tenderness and mild rigidity. Occurring in the right lower quadrant the syndrome may simulate appendicitis. Leukocytosis and polynucleosis are either slight or absent. Most cases of this type occur in miliary infections of infants.

**2 The Fibrinous Form**—A strictly dry fibrinous type rarely occurs. There is generally a preponderance of fibrinous exudate with a minimal amount of effusion. The onset is more insidious than in the ascitic form and fever is generally slight or absent. Localized collections of fluid may form soft doughy masses and the abdomen may become symmetrically or irregularly enlarged. Contracting adhesions may produce mechanical ileus or cause circulatory disturbances and edema with resulting digestive upsets and renal insufficiency. Occasionally the fibrinous form is a sequel of the ascitic.

**3 Tabes Mesenterica Form**—Many mild cases of tabes mesenterica occur asymptotically and are unrecognized clinically. In the average type there is a moderate generalized adenopathy of the mesenteric nodes. The condition may occur alone or be associated with the other forms. In severe infections the glands caseate and produce soft nodular masses (cold abscesses).

**4 Ulcerative or Caseous Form**—This is the most serious type and its occurrence is an evidence of inadequate resistance. The pathology may occur as a primary condition, develop in the progress of other forms, or be associated with tuberculosis elsewhere, especially pulmonary tuberculosis. Abdominal pain and tenderness are generally the first symptoms. The nodular foci soon caseate and produce large doughy purulent masses which may ulcerate into the intestine or be evacuated through the abdominal wall. Secondary infection may result. In severe cases the clinical picture is one of

sepsis intermittent pyrexia rapid pulse emaciation and progressive fatal toxemia

**Diagnosis**—Tuberculous peritonitis is the dominant cause of ascites in children. In conjunction with a doughy abdomen nodular masses slight evening fever and a tendency to diarrhea tuberculosis is highly probable. Chest roentgenograms frequently exhibit a widening of the mediastinal shadow from enlarged tracheo-bronchial nodes but pulmonary lesions are uncommon. Repeated negative tuberculin reactions exclude tuberculosis except in severe cases in which the response may be lost; a positive reaction indicates the presence of tuberculous somewhere in the body but not necessarily in the peritoneum. Leukocytosis and polynucleosis are absent.

Ascites in cardiac and renal disease is associated with dropsy elsewhere. In Banti's disease the liver and spleen are enlarged and anemia is more pronounced. Ovarian or dermoid cyst produces a focal mass often palpable by rectal examination and shifting dullness is absent. Chronic peritonitis should always be considered *tuberculous unless proven otherwise*.

**Prognosis**—This varies with the age of the child, type of lesion, dissemination elsewhere in the body and particularly with the patient's resistance. The younger the child the worse is the prognosis. In the ascitic form the fluid may absorb and the tubercles disappear; residual adhesions however are likely. Fibrinous types generally pursue a more chronic course, tend to recover but leave firm adhesions. Slight relapses are not uncommon. As previously stated, mild cases of *tuberculosis mesenterica* may fibrose and calcify without clinical recognition. Many average cases recover. Caseous forms are extremely serious. They generally pursue an acute course and are often complicated by ulcerative enteritis and mixed infection. Death may result from sepsis, intestinal obstruction, fecal fistula or disseminated tuberculosis.

Tuberculous complications may also develop in other organs: the pleura, pericardium, meninges, lungs or intestines. The kidneys are seldom involved in early life. It should be emphasized that only a small proportion of deaths are due to tuberculous peritonitis *per se*. The majority result from tuberculosis elsewhere, especially pulmonary.

**Medical Treatment**—The essentials of medical therapy for any tuberculous process comprise a hygienic dietetic regimen combined with abundant heliotherapy and complete rest. (Refer to Tuberculous Cervical Adenitis.) Fever is no contraindication to a high protein intake. Hematinics are indicated for the secondary anemia and in severe cases repeated blood transfusions are valuable. Cod or halibut liver oil or their concentrates and viosterol are fortifying adjuncts. Roentgen therapy may be beneficial in the fibrinous types. If fluid is present it should first be withdrawn as the rays

are absorbed by it. Tuberculin therapy appears to be of greater value in tuberculous peritonitis than in any other form of the disease. The intraperitoneal injection of air, oxygen or nitrogen is not advisable as intestinal perforation may be caused thereby.

**Surgical Treatment**—Laparotomy is indicated in ascitic types which do not respond favorably to medical treatment. Simple evacuation of the fluid is the usual procedure, the theory being that the resulting hyperemia of the peritoneum produces a beneficial effect in promoting regression and absorption of the tubercles. At the time of operation, however, a primary focus should always be sought in the appendix, cecum or Fallopian tubes. The removal of such nidus greatly enhances the prospect of recovery. Drainage is contraindicated except for suppuration.

In fibrinous types without much ascites, laparotomy is less effective. It is nevertheless justifiable for in many instances the primary focus may be removed. Enucleation of enlarged glands in tubal mesenteric types is contraindicated. Injury to the mesenteric vessels may result thereby and cause necrosis of the gut.

Intestinal obstruction due to bands, kinks and ulceration may require operative interference. Great care must be exercised in separating adhesions as the bowel is readily perforated. Soft fluctuating masses (cold abscesses) should be undisturbed unless they produce urgent symptoms. Evacuation and drainage may result in dangerous mixed infection eventuating in sinus formation or fecal fistula. Sinuses should be swabbed with 95 per cent phenol followed by tincture of iodine. Injection with Beck's paste is at times beneficial. Silver nitrate should be avoided.

### GONORRHEAL PERITONITIS

Despite the frequency of specific vaginitis in infancy, gonococcus invasion of the peritoneal cavity occurs very seldom. When the infection ascends in the genital tract the Fallopian tubes generally become sealed and thereby limit the process.

**Pathology** The peritoneum is involved secondarily from either salpingitis or pyosalpinx. The Fallopian tubes are red and thickened and creamy odorless pus may often be expressed from their fimbriated ends. The inflammatory process tends to localize in the pelvis and the intestinal coils become bathed and matted in the exudate. Although the lower abdomen may also become involved, diffuse peritonitis rarely develops. Mixed infection (symbiosis) seldom occurs except in chronic cases.

The purulent exudate may become absorbed or produce a pelvic abscess and the salpingitis may resolve or eventuate in pyosalpinx. Extensive adhesions may be the ultimate residuum and future sterility may result from tubal incompetence. The gonococcus is

usually demonstrable in the cervical and urethral smears. Cystitis is a common complication.

**Symptomatology**—Following an indefinite period of vaginitis invasion of the peritoneal cavity produces sudden pain, vomiting, hyperpyrexia, leukocytosis and frequently bladder irritability. The pain is often intense. Although the tenderness and muscular rigidity or hyperesthesia of the skin may be generalized at first, the signs soon become confined to the lower abdomen. Rectal examination elicits acute tenderness in the early stages. Distention occurs slowly and is seldom marked.

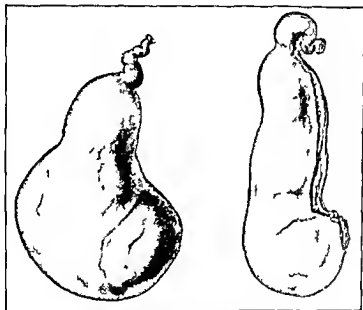


FIG. 17J. B. lateral pyosalpinx in a child aged 12 years.

The acute symptoms usually subside in a few days and the condition may resolve or eventuate in pelvic abscess. Adhesions form commonly between neighboring intestinal coils and relapses are common.

**Diagnosis**—The differential diagnosis from perforative appendicitis may be extremely difficult in young children. (Refer to Appendicitis.) Positive smears are strongly suggestive. In doubtful cases an exploratory laparotomy should be performed. If the pathology is found to be gonorrheal, drainage is unnecessary. Whereas acute salpingitis should be undisturbed, a definite pyosalpinx requires salpingectomy.

**Prognosis**—Death rarely occurs from gonorrheal peritonitis. Sterility, however, is a common sequel.

**Treatment** Expectant treatment in the Fowler position should always be employed. Most cases eventuate in resolution. Occasionally a pelvic abscess requires evacuation and drainage and infrequently a pyosalpinx necessitates removal (Fig 179). Surgery may also be required in cases which develop chronic intestinal obstruction.

### PNEUMOCOCCUS PERITONITIS

*Pneumococcus peritonitis* is of uncommon occurrence. It is predominantly a disease of early childhood and chiefly affects girls under ten years of age.

**Etiology** Present opinion doubts the actual occurrence of primary peritoneal invasion by the pneumococcus. Diligent search will usually reveal a primary nidus most commonly in the Fallopian tubes, less often in the lungs or pleura and infrequently in the middle ear. At times the infection occurs simultaneously in the lungs and peritoneum from pneumococcus septicemia. Blood cultures are often positive especially in the early stages.

Approximately 70 per cent of the cases occur in girls between the ages of three and seven years. Pneumococci of the same type have repeatedly been isolated from the peritoneum, vaginal tract and blood. The peritonitis is generally most severe in the pelvis and apparently results from an ascending primary vaginal infection. Experimental pneumococcus inoculation of the vaginal tract of female monkeys produces an identical type of peritonitis from ascending infection through the uterus and tubes.

**Pathology** Cases due to tubal infection begin as a pelvic peritonitis which often becomes rapidly diffuse. The maximum pathology is evidenced in the pelvis and lower abdomen and at times pus may be made to extrude from the tubes upon stripping them. In the early stages the exudate is a thin milky or flaky pus of grayish yellow or greenish color and without odor. The moderately injected peritoneum exhibits a slimy slippery feel and there is little tendency to the formation of adhesions. Mesenteric lymphadenitis is often pronounced. At a later stage the pus becomes thicker and contains abundant fibrin clots and plastic strings. Delimiting adhesions result therefrom and large abscesses may develop. The latter if untreated may evacuate themselves spontaneously at times through the umbilicus. Bacteremia is generally demonstrable in severe cases. Enteritis is a rather constant complication and pneumonia, pleurisy or pericarditis may be coexistent.

**Symptomatology** The sudden onset of abdominal pain and vomiting accompanied by severe toxemia, hyperpyrexia, high leukocytosis and polynucleosis is characterized by the absence of a primary focal causative lesion. Toxic diarrhea also occurs in most cases. The temperature is usually  $104^{\circ}$  to  $106^{\circ}$  F. and the leukocytosis is far greater than in ordinary suppurative peritonitis, counts of 30,000 to 40,000 being quite common with polynucleosis above

90 per cent. Marked distention occurs early, tenderness may be less evidenced than skin hyperesthesia and the abdomen often feels more doughy than rigid.

Fulminant cases may succumb to pneumococcus sepsis in a few days. In less severe infections a remission of general symptoms usually occurs in five to seven days but the diarrhea and abdominal distention may persist for weeks and the disease pursue a rather chronic course. Complete resolution may follow. In other instances, a fluctuant swelling develops in the subumbilical region which requires evacuation.

**Diagnosis**—Indefinite physical signs may render the diagnosis extremely difficult. High leukocytosis and polymucleosis are suggestive. Hyperpyrexia occurring within a few hours is very unusual in appendicitis. In the absence of focal signs, the occurrence of diffuse peritonitis in female children should strongly suspicion pneumococcus or gonococcus infection.

The diagnosis may be verified by vaginal smears and cultures or by peritoneal puncture made in the mid-line, 1 inch below the umbilicus, with a short bevel No 16 gauge needle. Agglutination tests with the patient's serum may give positive results as to the pneumococcus type. This is of value if serum treatment is favored.

**Prognosis**.—Statistical reports exhibit wide variations. Except in mild infections, the prognosis is grave and the mortality probably exceeds 80 per cent.

**Treatment**—The rational therapy of peritonitis is based upon two cardinal factors—the removal of the cause and the relief of intraperitoneal pus tension. When the causative pathology cannot be removed, it is questionable whether operation is advisable, the drainage of diffuse peritonitis is quite impossible and drains by acting as foreign bodies may continue a peritonitis which would otherwise subside. Cases in which the pneumococcus peritonitis is a bacteremic sequel of pneumonia, pleurisy or otitis media are therefore probably best treated by supportive measures and absolute peristaltic rest in the Fowler posture. (Refer to Acute Suppurative Peritonitis.)

**Indication for Operation**—When pneumococci are present in the vaginal smears or cultures, the abdomen should be opened in the lower mid-portion and bilateral salpingectomy performed if definite salpingitis is demonstrable. The exudate is then aspirated and the abdomen closed without drainage. Surgical intervention should be delayed, however, in cases of rapid ascending infection which exhibit rapid pulse and respiration, stupor or delirium, and especially slight cyanosis.

Localized accumulations of exudate should be incised and drained. In selected types, serum treatment appears to be advantageous and in protracted cases repeated blood transfusions are especially beneficial.

## POSTOPERATIVE PERITONEAL ADHESIONS

The gentlest handling of tissues is an absolute requisite of good pediatric surgery and in no part of the body is this more essential than within the peritoneal cavity. Undue handling and exposure of the viscera dry sponging dry laparotomy pads washing with saline or antiseptics and the indiscriminate insertion of drains are all definitely damaging to the delicate endothelium. Fibrin appears rapidly at the site of insult and during the period of postoperative freedom from peristalsis agglutination may become well established and eventuate in fibrous organization.

Whereas early restoration of peristalsis is favorable to the separation of agglutinated surfaces leukocytic ferments are the chief agents in the absorption of fibrinous exudate. Although newly formed adhesions tend to spontaneous regression in certain patients there is an inherent tendency to fibrosis.

Visceral adhesions only produce symptoms through the mechanical interference of mobile or hollow propulsive organs. parietal adhesions may also cause pain when pulled upon. There is little relationship however between the extent of adhesions and the symptoms produced thereby. Asymptomatic adhesions should be left undisturbed as new ones of greater density may reform.

**Prophylactic Agents**—The prophylactic virtue of foreign substances such as oils waxes and membranes is nil they may actually produce adhesions. The recent therapy of instilling amniotic fluid into the peritoneal cavity in cases of postoperative adhesions offers promise of benefit. Its use is contraindicated in the presence of active peritonitis.

**Symptomatology** The dominant early symptom is intestinal obstruction. This generally occurs insidiously during the second week of convalescence by which time the peritonitis has subsided. There is progressive distention accompanied by vomiting and finally a failure to pass flatus or feces with enemas or irrigations. Visible peristalsis seldom occurs. The vomiting of upper intestinal contents is conclusive and ominous. Unless relief is obtained promptly by gastric lavage or duodenal suctionage through aid of the Levine tube early operative interference becomes imperative.

**Diagnosis** The differential diagnosis from paralytic ileus (dynamic) is often difficult. The latter usually occurs in the first few postoperative days during the stage of active peritonitis. Both the large and small intestines are involved and the abdomen is conspicuously silent. Mechanical (dynamic) obstruction is usually confined to the small intestine and auscultatory swishing and borborygmi may be heard at the site of obstruction.

Careful wound inspection should be made in all cases of suspected obstruction as dehiscence is particularly common in children. Although the skin sutures remain intact an exudate of sanguineous



serum usually indicates wound gapping associated with intestinal or omental protrusion. In such cases the patient should be removed to the operating room and the wound thoroughly explored.

Intestinal obstruction from adhesions may also occur months or years after operation and the onset may be gradual or acute. In the presence of distention, vomiting and failure to pass gas or feces the diagnosis of obstruction is self-evident. Visible peristalsis is often exhibited in these types. In subacute cases with partial obstruction roentgen ray examination may locate the site of pathology. It should be emphasized that mild fibrotic tuberculosis of the peritoneum is a more common cause of intestinal obstruction than is generally recognized. Some years ago the writer operated upon a girl aged twelve years for recurrent intestinal obstruction. There had been three previous laparotomies during a two-year interval. The first was for subacute appendicitis and the subsequent two for intestinal obstruction. At operation the terminal ileum was found sharply kinked by pelvic adhesions and both Fallopian tubes exhibited minute tubercles. Following bilateral salpingectomy the child has remained well.

**Treatment** Unless relieved by palliative measures prompt surgical intervention becomes imperative. Obstructing bands, kinks or adhesions should be carefully revised. Rough separation of the latter may result in perforation. Denuded areas should be peritonealized when possible and amniotic fluid may be instilled into the peritoneal cavity before closure. Temporary high ileostomy has been largely replaced by duodenal suctionage through employment of the Levine tube.

The abdominal wall should be carefully closed in layers, preferably with chromic catgut and reinforced with non absorbable retention sutures. Proper maintenance of an adequate water-saline balance is imperative. (Refer to Dehydration.)

## TUMORS AND CYSTS OF THE PERITONEUM

Tumors of the peritoneum are very rare. They may be primary or secondary and benign or malignant. The reported cases comprise fibroma, angioma, lipoma and sarcoma. Lipoma is usually secondary to growths arising in the subperitoneal fat and sarcoma to retroperitoneal growths.

Cysts of the peritoneum are of unusual occurrence and are found chiefly in the mesentery. Embryonal types may develop from remnants of the vitello-intestinal duct, the Wolffian body or from fetal inclusions (dermoids). Parasitic cysts are usually echinococcal and rarely cysticercic. Serous cysts may result from the localization of inflammatory exudate and chylous cysts from mesenteric lacteal blockage.

## CHAPTER XXXII

### THE LIVER AND BILIARY SYSTEM

DURING early life the liver is relatively large and its edge is normally palpable up to five years of age

#### ABNORMALITIES OF THE LIVER AND BILIARY SYSTEM

In the rare instances of situs inversus of the abdominal organs the liver is transposed to the left side. Riedel's lobe is extremely uncommon.

The gall bladder may rarely be double or entirely absent. Occasionally it is completely surrounded by peritoneum and presents a variable length mesentery. The arrangement of the ducts is subject to considerable variation, whereas normally the bile duct opens into the duodenum in common with the pancreatic duct; each may have a separate ostium. The cystic duct at times parallels the course of the common hepatic duct for a considerable distance and in unusual instances an accessory right hepatic duct extends from the gall bladder fossa to join the common hepatic duct. Congenital obliteration of the ducts occurs with extreme rarity and in such cases they are replaced by fibrous cords.

#### PHYSIOLOGY OF THE LIVER

The functions of the liver are manifold and of those known many are incompletely understood. *Carbohydrates* In carbohydrate metabolism glucose is changed to glycogen. Although the latter is stored in both the hepatic and muscle cells, the liver is apparently the sole regulatory mechanism for blood sugar. *Proteins* The liver cells are necessary for the deamination of amino-acids into ammonia and urea and for the synthesis of uric acid. It appears that dextrose may also be formed in small amounts from amino-acids and lactic acid. *Fats* The bile salts are intimately associated with the oxidation of fats and possibly with lecithin formation.

**Metabolism of Biliary Constituents**—The chief metabolic constituents of bile are bile salts, cholesterol and bilirubin. The value of bile salts both as a cholagogue and an aid in the saponification and absorption of fats is well recognized. Their actual formation however is incompletely understood and the metabolism of cholesterol is unknown. Bilirubin is formed chiefly in the bone-marrow and to a lesser extent in the spleen and liver. Thus, in respect to

bilirubin the liver is mainly an excretory organ and various tests of hepatic function are based thereon

**Detoxifying Function**—The liver possesses powerful detoxifying function. Microorganisms in the blood stream are removed in large numbers and are partly excreted in the bile. This is especially evident in typhoid fever and this mechanism may account for many cases of cholecystitis. Particulate matter and mineral poisons such as arsenic mercury and phosphorus are sequestered and acted upon by the Kupffer phagocytic cells. Response to continuous infection may result in hepatitis and cirrhosis.

**Coagulation of the Blood** Whipple and Hurwitz demonstrated that hepatic injury in dogs causes a marked lowering of the fibrinous level in the blood plasma. The findings of other investigators upon antithrombin and calcium metabolism further indicate that the liver may be a definite factor in the little understood function of blood coagulation.

**Tests of Hepatic Function** The only tests of clinical value are certain procedures which estimate the injury to the bile excretory mechanism. These comprise the dye retention tests of Rowntree and Rosenthal, the Van den Bergh reaction for retained bile pigment and the coagulation time of the blood. The sugar tolerance tests are of little if any value.

**Rowntree Rosenthal Test** In this technic a measured quantity of the dye according to kilogram weight is injected intravenously and the rapidity with which the dye is removed from the blood is estimated at different intervals by colorimetric methods. Retention of the dye however is not always associated with bilirubin retention and the test is therefore of only relative value.

**Van den Bergh Test**—This applies the diazo-reaction to the blood serum and is a delicate test for latent jaundice. Bilirubin normally occurs in the blood in the proportion of 1 to 400 000 to 1 to 1 000 000. The unit employed is 1 to 200 000. A prompt direct reaction occurs in obstructive jaundice; an indirect reaction suggests a hemolytic or toxic origin. The test is especially valuable in detecting latent jaundice and in estimating its increase or subsidence. The interpretations of the finer reactions are subject to error.

**Coagulation and Clotting Times**—These are instructive but do not always give an accurate index of what will occur during operation or subsequently.

**Cholecystography (Graham Cole)** This is a valuable aid in the diagnosis of gall bladder disease. It is dependent upon the fact that phenoltetraradophthalein is eliminated in the bile, collects and becomes concentrated in the normal gall bladder and produces a roentgen shadow. Normally a faint shadow appears in four hours, a deeper one in eight and a dense but somewhat smaller shadow in twenty-four hours. By thirty-six hours the shadow generally dis-

appears. A delayed or imperfect shadow indicates insufficient concentrating power due to disease of the gall bladder or impaired eliminative function of the liver or obstruction of the ducts which prevents bile from entering the gall bladder. Gall stones are demonstrable in about 75 per cent of cases.

The foregoing tests are distinctly helpful in diagnosis, all are subject to a percentage of error, however, and complete dependence should not be placed upon them. The Van den Bergh reaction is extremely valuable in detecting latent jaundice and is the only test commonly employed in childhood.

## JAUNDICE

### (ICTERUS)

Jaundice is a symptom and implies an abnormal amount of bile in the blood and all body tissues imparting to the latter a characteristic yellow color. It is of twofold significance to the surgeon: (1) From the standpoint of differential diagnosis, and (2) the increase of surgical risk in its presence. The term latent jaundice is employed to describe a condition of bile in the blood stream which is not evidenced by clinical symptoms.

**Metabolism of Bilirubin.** Bilirubin is derived from hemoglobin. Although the cells of the entire reticulo-endothelial system are probably capable of destroying worn-out erythrocytes and disintegrating their hemoglobin, the process is concentrated in the liver, spleen, bone-marrow and lymph nodes. The bilirubin thus formed is carried to the liver and on passing through the polygonal cells before entering the bile apparently undergoes some definite change the nature of which is not understood. The direct and indirect Van den Bergh reactions are predicated upon this difference occurring in bile that has passed through the liver cells and that which has not.

**Classification of Jaundice.** The modern concept of jaundice is based upon the almost proven fact that the polygonal cells of the liver do not manufacture bilirubin but rather transfer it from the vascular capillary system to the biliary channels and in this transfer modify the pigment. Predicated upon this hypothesis and also for practical clinical purposes, jaundice cases may be divided into three main groups: *obstructive*, *infectious* and *hemolytic*. Many cases, however, belong to more than one type. Jaundice of the new born will be first described.

**Icterus Neonatorum.** This type does not concern the pediatric surgeon. Careful observations indicate that slight jaundice occurs in almost all new born infants. Whether it is of hepatogenous or hematogenous origin remains controversial. Polycythemia normally present at birth soon disappears through the destruction of

excessive erythrocytes. Such hemolysis appears to be the basic factor in the production of *icterus neonatorum*. The indirect Van den Bergh reaction also suggests an extrahepatic origin.

Clinical jaundice occurs in 10 to 20 per cent of the new born. It usually appears on the second or third day and affects chiefly the skin and to a lesser degree the sclera and mucous membranes. It is entirely asymptomatic except at times the babies are unduly sleepy, the temperature and pulse are unaffected, the stools are normal and bile pigment is not demonstrable in the urine by the ordinary tests. The jaundice generally lasts only a few days but in unusual cases may continue two weeks.

**Diagnosis** —The differential diagnosis from *symptomatic jaundice* especially that due to sepsis is made upon the absence of a presumptive cause. In general early slight discoloration favors *icterus neonatorum* and a later more intense jaundice some other etiology. Congenital *atresia of the bile ducts* produces jaundice promptly after birth and the condition is one of persisting increased severity. The stools are acholic and the urine is dark colored from excessive urobilin. The anomaly causes death within a fortnight.

**Obstructive Jaundice** —*Catarrhal icterus* is the dominant type of obstructive jaundice in early life. The disease is decidedly more common in older children than in infants and is generally a sequel of gastroduodenitis. Congestion of the duodenal mucosa with involvement of the papilla of Vater may not be the etiologic factor in all cases however for at times mild epidemics of icterus occur which suggest an infection of the bile ducts or possibly of the liver cells. Duct obstruction from biliary calculi, new growths and pancreatic disease are pathologic rarities in early life.

**Symptomatology** —The characteristic yellow discoloration of the skin, mucous membranes and urine usually follows a mild gastrointestinal disturbance. The icterus becomes well developed in twenty four to forty eight hours and is often associated with slight pyrexia, abdominal fullness and mild hepatic tenderness. Headache, malaise and anorexia are commonly present and in severe cases there may be itching of the skin and bradycardia. The urine contains an excess of urobilin and the stools are light colored or acholic. The icterus usually fades within ten days and seldom lasts over three weeks. The Van den Bergh reaction is direct after the icterus appears although occasionally it is indirect in the prodromal stages.

**Treatment** —This comprises a fat free diet, abundant fluids and an occasional saline laxative. Duodenal drainage with Epsom salt may shorten the duration. The disease is self limited and eventually enters in recovery. The surgeon is rarely consulted except in protracted cases.

**Infectious Jaundice** —In these types the icterus is dependent upon actual damage to the liver parenchyma. The bilirubin may not

only fail of excretion but that which is excreted may be reabsorbed. Accordingly the Van den Bergh reaction may be direct indirect or diphasic. Many cases of catarrhal jaundice especially of the epidemic type fade imperceptibly into this category. The actual pathology of the angiocholitis may be a choking of the minute intra-hepatic vessels from released blood coloring matter or clogging of the bile capillaries from congestion or viscid bile. The condition is a common complication of infectious diseases such as pneumonia scarlet fever typhoid fever malaria and especially sepsis. Infectious jaundice may also occur with parasitic or spirochetal infections of the liver and from certain drugs. Only cases complicating sepsis concern the surgeon.

**Hemolytic Jaundice**—In hemolytic jaundice there is an excess amount of bilirubin (or its precursor) and as it has not passed through the polygonal hepatic cells the Van den Bergh reaction is delayed or indirect. The cases are characterized by the following: the absence of bile pigment in the urine the presence of urobilin ample color to the stools increased fragility of the erythrocytes, and not infrequently splenomegaly.

The surgeon is occasionally consulted in cases of congenital and familial hemolytic jaundice. In *congenital hemolytic jaundice* the icterus is present at birth or develops soon thereafter and simulates the severe type of icterus of the new born (icterus gravis). The jaundice is intense and may be persistent. Some cases are accompanied by a hemorrhagic tendency especially of the skin mucous membranes and umbilicus. The stools are yellow and at times bloody the urine contains urobilin and frequently bilirubin. The prognosis is largely influenced by the element of hemorrhage.

*Familial hemolytic jaundice* probably results from some defect in the blood forming process plus an increased destruction of erythrocytes in the spleen. The icterus may be congenital appear some time after birth or not until childhood or adult life. The jaundice is seldom intense and varies in degree from time to time when once established. The urine is dark colored from urobilin but free from bilirubin and the feces contain bile. The spleen is constantly enlarged and an increased fragility of the erythrocytes is generally demonstrable. The blood exhibits secondary anemia with a few megaloblasts normoblasts and numerous reticulated red cells. Leukocytosis is not evidenced. A hemorrhagic tendency may accompany the disease.

The patients may live for years the jaundice never quite disappearing. Exacerbations are common especially after gastro-intestinal upsets and chilling. Some patients enjoy fair health despite the constant mild jaundice.

**Diagnosis**—This is made from the familial history, mild persistent jaundice the absence of bile in the urine its presence in the feces

increased fragility of the red corpuscles and splenic enlargement Banti's disease is predominantly a splenic disorder the biliary cirrhosis with jaundice appears late the icterus is progressive and more intense there is no increased corpuscular fragility and a familial history is absent

**Treatment**—Although cases of familial hemolytic jaundice have been definitely benefited by splenectomy operative interference should never be attempted unless the disease is progressive and the patient is suffering The degree of improvement following splenectomy is problematic

### ASCITES

The condition is rarely congenital and seldom develops during infancy Cardiac decompensation nephritis and tuberculous peritonitis are the usual etiologic factors others comprise (1) increased pressure in the portal circulation from hepatic or pulmonary disease (2) extrahepatic pressure on the portal veins from tumors enlarged mesenteric glands lymphatic leukemia malignant lymphomas parasitic cysts or splenomegaly (3) polyserositis and (4) pernicious or very grave secondary anemia

**Varieties of Ascites**—(1) *Serous* The fluid is clear amber color the specific gravity in transudates being 1.010– and in exudates 1.015+ (2) *Hemorrhagic* This may occur in tuberculosis volvulus and intussusception (3) *Chylous* The condition results from lacteal or thoracic duct block

**Symptomatology**—The abdomen is symmetrically enlarged and at times enormously distended The skin becomes tense and shiny and the umbilicus may protrude The characteristic signs of free fluid are readily demonstrable—fluid wave and shifting dullness The condition must be differentiated from tympanites ovarian and dermoid cysts distended bladder and large abdominal tumors

**Treatment** Ascites is but a symptom Therapy concerns the provocative disease

### CIRRHOSIS OF THE LIVER

Cirrhosis of the liver is rare in childhood and exceedingly so in infancy In the portal type (Laennec's cirrhosis) the connective-tissue fibrosis compresses and penetrates the lobules the organ becomes reduced in size and the partial portal obstruction leads to ascites In the hypertrophic variety (Hanot's cirrhosis) the interlobular overgrowth of connective tissue compresses the biliary ducts the resulting jaundice is associated with hepatic and splenic enlargement

Too much emphasis however has been given to the factor of hypertrophic and atrophic types In the early inflammatory stages

of cirrhosis, the liver tends to enlarge, and in the final process of fibrosis, shrinkage occurs. In the hypertrophic form, in addition to the jaundice which may be intense, bilirubin occurs in the urine, the spleen is greatly enlarged and ascites is variable. In the atrophic type, ascites is the dominant symptom and jaundice is absent or only slight. Gastric hemorrhages may also occur and leukocytosis is frequent.

**Diagnosis.**—Familial hemolytic jaundice is distinguished by the absence of bile in the urine, the absence or only slight degree of liver enlargement, and definite increased fragility of the erythrocytes. In Banti's disease the predominating symptom is early splenic enlargement; jaundice is less marked and leukopenia is present.

### CYSTS OF THE LIVER

Echinococcus cysts of the liver are rare, multiple small congenital cysts have also been described, in some instances associated with bilateral polycystic disease of the kidneys.

**Echinococcus Cyst (Hydatid Cyst).**—Cysts due to the *Tenia echinococci* occur most frequently in the liver; the hooked embryo, after penetrating the intestinal wall, is carried thence by the portal radicles. The cysts develop slowly and painlessly, may attain large size, and are surrounded by a zone of connective tissue. Their walls consists of an outer elastic capsular layer (ectocyst) and an inner germinal (endocyst) from which multiple scolices develop. Secondary or daughter cysts may arise from the endocyst either within or without the cyst capsule. The clear water content is of low specific gravity, free from albumin and contains myriads of hooked embryos.

Whereas in some instances the tenia may die and the cyst shrink and calcify, in most cases the cyst continues to enlarge and rupture may occur into neighboring organs: the peritoneum, stomach, intestines, vena cava, pleura, pericardial sac or lungs; more frequently, however, suppuration develops and the disease pursues a severe septic course.

**Symptomatology.**—The gradual enlargement of the liver is often accompanied by dull aching pain. Tenderness and jaundice are absent and the condition remains afebrile unless suppuration occurs. If the cyst develops in the lower portion of the liver, a rounded fluctuating mass with hydatid thrill may be elicited. Hepatic roentgenograms often exhibit the globular mass clearly outlined. Eosinophilia is constantly demonstrable. The Casoni intradermal and complement-fixation tests are specific diagnostic aids.

**Treatment.**—A few cures have been reported following the intravenous injection of arsphenamine. When possible, the entire cyst should be removed *en masse*. Remaining portions of the endocyst,



daughter cysts or scolices will produce recurrence and dissection frequently accompanied by serious secondary infection.

The operation should be performed in two stages—by either a transabdominal or transthoracic approach. In the former the cyst wall or liver covering it is exposed and the wound is packed with gauze for seventy-two hours to promote adhesions. In a necessitated transpleural approach the eleventh and twelfth ribs are resected and the diaphragm is sutured to the parietal wall to prevent subsequent contamination of the pleural cavity. After an interval of three days or longer an attempt is made to remove the entire cyst after first injecting it with tincture of iodine, ether or 1 per cent formalin to kill the embryonic scolices. If enucleation is impossible the major cyst portion including the endocyst lining is removed and the cavity marsupialized. Through subsequent infection the sac becomes spontaneously separated from the liver and is withdrawn. Recurrence may occur even after many years.



FIG. 180.—Primary carcinoma of the liver.

### TUMORS OF THE LIVER

Tumors of the liver are of exceptional occurrence. The benign growths include congenital uni- and multilocular cysts, angioma, fibroma, lipoma, and adenoma. Large cheesy tumefactions occasionally develop from tuberculosis.

The malignancies comprise carcinoma, adenocarcinoma, and sarcoma, and Woolstein describes a primary epithelial tumor

**hepatoma** Although the majority of hepatic tumors are secondary metastatic manifestations primary carcinoma appears to occur more often in the liver than in any other organ in early life and over 100 cases have been reported (Fig 180)

### INJURIES OF THE LIVER

The liver is frequently injured through kicks blows and especially *crushing in runover accidents* also by the jagged ends of fractured ribs and by stab and bullet wounds Traumatic rupture usually involves the right lobe particularly its convex surface

Minor injuries may produce contusions of the liver without tearing of the capsule when more severe the superficial portion of the organ may be lacerated together with a rent in Glisson's capsule or a central laceration may be produced resulting in a hematoma and possible subsequent cyst or abscess formation In severe trauma the liver may be pulped Penetrating wounds may cause profuse hemorrhage

**Treatment**—The symptoms of severe liver damage comprise those of shock and intra abdominal hemorrhage Immediate laparotomy should be performed and the bleeding controlled by pressure hot pads packing cautery coagulation or suture Transfusion of whole blood at the time of operation is often life-saving When unavailable 10 per cent glucose in physiologic saline should be administered intravenously In many cases of profound shock packing alone is all that can be done Bleeding from the portal radicles is readily controlled thereby as the portal pressure does not exceed 30 mm. of mercury

Following operation the patient should be returned to bed in the shock position and be surrounded by warm blankets and hot water bottles The lower extremities may be bandaged from below upward to conserve the depleted circulation and a sand bag may be placed over the abdomen to prevent splanchnic engorgement Ample saline solution should be administered by phleboclises or hypodermoclises Subsequent blood transfusions are specifically supportive Patients who survive the primary shock usually recover for the liver is endowed with great reparative potential

### ABSCESS OF THE LIVER

Suppurative hepatitis is relatively infrequent in early life The condition usually results from parasitic or pyogenic infection and rarely from trauma or foreign bodies The abscess may be solitary or multiple

**Amebic Abscess**—Amebic or tropical abscess develops as a complication or sequel of amebic dysentery through hepatic invasion

by the *Amœba histolytica*. The disease is endemic in the tropics and may be spread by carriers to distant parts.

**Pathology**—The process develops most commonly as a solitary abscess in the upper and posterior portion of the right hepatic lobe. The mass may gradually attain large size and ultimately rupture into the neighboring parts—through the diaphragm into the pleura, pericardium or lungs, into the stomach, intestines, peritoneal cavity or vena cava, or through the abdominal wall. The pus is chocolate-colored and generally contains amebæ.

**Symptomatology**—The sole symptom may be gradual hepatic enlargement. In conjunction with a previous history of amebic dysentery, this should always excite a strong suspicion of amebic abscess. Occasionally a definite bulging occurs. There may also be deep dull aching pain, or an unproductive cough may develop from phrenic nerve irritation. Not infrequently the earliest symptom is pleurisy. The condition is essentially afebrile and for a long period of time the general health may remain satisfactory. Roentgenologically, the liver exhibits asymmetrical enlargement.

**Prognosis**—In cases of unruptured solitary abscess the operative mortality approximates 75 per cent. Disseminated types are almost universally fatal.

**Treatment**—The patient should first be treated for amebiasis. Ipecac or emetin are usually prescribed, also treparsol, vioform or castela. Under vigorous medical treatment the amebæ may die and the abscess become sterilized. In some instances spontaneous recovery occurs, in others, aspiration may result in cure. The latter procedure, performed blindly through the chest or abdominal wall, is ill-advised. Dissemination may be produced thereby if the parasites are viable.

For similar reasons the abscess is best drained in two stages. The approach may be either transabdominal or transthoracic. In the former, the liver is exposed and walled off by gauze packing to promote adhesions, in the latter, two lower ribs are resected and the diaphragm is sutured to the parietal wall to exclude the thoracic cavity. After seventy-two hours and guided by an aspiration needle, the liver is incised, preferably by a cautery. Two soft rubber tubes are then inserted for drainage and irrigation. The discharge should be examined for parasites, if found present, further ipecac medication is indicated and the abscess cavity should be irrigated with quinine or emetin solution.

**Pyogenic Abscess**—Pyogenic bacteria are generally carried to the liver through either the blood stream (pyemia) or the portal vein (pyelphlebitis). Infection may also develop from penetrating wounds, hematoma following trauma, by direct continuity from peritonitis, perinephritic abscess or empyema, suppuration of hydatid cysts, miliary tuberculosis, the death of round worms

which may have entered the bile ducts or through invasion by the *Distoma hepaticum* or *Coccidium oviforme*.

**Pathology** Pyemic abscesses are usually small and multiple. Although several foci may fuse a solitary abscess rarely develops. The bacterial invasion occurring via the hepatic artery may be secondary to boils or furuncles, osteomyelitis especially of the cranial bones, ulcerative endocarditis, pyogenic pulmonary infections or general sepsis.

**Symptomatology**—Invasion of the liver is usually accompanied by a chill followed by hyperpyrexia. The subsequent course is that of sepsis—intermittent or remittent fever with wide temperature excursions, chills and sweating. The focal signs are not marked; the liver gradually enlarges and may become tender especially upon deep percussion and slight jaundice usually develops. Leukocytosis and polymorphonuclear leukocytes are moderate and bacteriemia may or may not be demonstrable. The progressive toxemia is almost universally fatal.

**Treatment** A solitary abscess when demonstrable should be incised and drained. This rarely occurs and the treatment is necessarily limited to supportive measures. Frequent small transfusions of whole blood are perhaps more effective than larger amounts administered less often. Autogenous vaccines and bacteriophages are at times beneficial. The intravenous administration of germicides is not recommended.

### PHLEPHLEBITIS

Purulent phlebitis of the portal veins may follow severe infection in areas drained by the portal radicles. Acute appendicitis especially the gangrenous type accompanied by septic thrombosis is the dominant cause. Clumps of bacteria or thrombotic segments are carried via the appendiceal, ileocolic and superior mesenteric veins to the portal vein and thence to the liver with resulting metastatic abscess formation. Externally the portal vein may appear normal yet its intima may be studded with multiple thrombi. In rare instances phlephlebitis may also result from diverticulitis or infections in the spleen, pancreas or rectum. The predominant organism is *B. coli*.

**Symptomatology** The complication of phlephlebitis in acute appendicitis usually develops within seventy-two hours and rarely after the first week. The onset is associated with a severe chill and the process pursues a septic course—intermittent or remittent hyperpyrexia, recurrent chills and sweating. The progressive toxemia is accompanied by an increasing pulse rate and gradual enlargement of the liver. The edge of the latter is often tender and deep percussion of the organ may cause pain.

The abdomen may remain soft or become somewhat distended and the spleen is frequently palpable. Jaundice is variable, bilirubin is increased in the blood serum and an indirect Van den Bergh reaction is usually demonstrable. The degree of leucocytosis and polynucleosis is inconstant and inconsequential. Bacteriemia is uncommon.

**Prognosis** The prognosis is exceedingly grave and practically all cases succumb to sepsis, cholemia or bronchopneumonia within six weeks.

**Treatment** — Pylephlebitis probably causes 5 per cent of the deaths from acute appendicitis and prompt surgery in the latter is the only safeguard. If the appendiceal and ileocolic veins are found thrombosed at the time of operation they should be opened and drained. Ligation of the portal vein is futile. The treatment of metastatic abscesses within the liver is necessarily supportive (Refer to Pyogenic Abscess).

## DISEASES OF THE GALL-BLADDER AND BILIARY SYSTEM

Acute cholecystitis and acute cholangitis seldom develop in early life except as a complication of typhoid fever or sepsis. In rare instances intestinal protozoa invade the biliary ducts and the *Ascaris lumbricoides* may enter the common duct and cause inflammation.

Chronic cholecystitis is a rather frequent sequel of typhoid fever. The condition is perhaps more common than is generally recognized but the pathology seldom produces symptoms before adult life. Tuberculosis of the gall bladder is extremely rare and involvement of the ducts even more so.

Gall stones are very infrequent in children. Although they may produce the same clinical picture as in adults the symptoms are frequently attributed to disease of a high situated appendix. The author made this error in a girl aged fifteen years. The literature concerning gall bladder disease in early life is so scant that case records merit reporting.

## CHAPTER XXXIII

### THE SPLEEN AND PANCREAS

#### THE SPLEEN

EXCEPTING traumatic lesions splenic surgery is chiefly associated with splenomegaly and allied diseases of the blood. Although based largely upon empiricism the therapeutic value of splenectomy has been definitely established in certain rather well-defined conditions. In the subsequent discussion only those pathologies which are amenable to surgery will be stressed.

#### ANATOMY OF THE SPLEEN

The capsule of the spleen or tunica albuginea is composed of firm connective tissue containing both elastic and smooth muscle fibers. It is surrounded by a peritoneal envelope the tunica serosa. Septa and trabeculae pass inward from the tunica albuginea and divide the organ into lobes and lobules. Each of the latter is subdivided by fine trabeculae into ten or more lesser anastomosing compartments which comprise the splenic units (Fig 181). These are filled with splenocytes or pulp cells apparently supported upon a fine reticulum and constitute the splenic reticulo-endothelial system. The cells appear to be definitely phagocytic for both erythrocytes and particulate matter.

The blood vessels course the trabeculae. Upon entering the central portion of the lobule the arteries acquire an additional fibrous sheath the latter thickens and contains small cells with large nuclei the Malpighian bodies or lymph follicles. The terminal vessels to each lobular unit traverse the splenic pulp and end abruptly in globular masses of cells termed ellipsoids. From the end capillaries blood flows through the pulp spaces seeping through the openings in the walls of the numerous sinuses into the collecting veins. The intercellular pulp spaces are minute and the ellipsoids in addition to being actively phagocytic may act as filters of particulate matter.

The Malpighian bodies or lymph follicles numbering 10 000 or more vary in size up to 0.6 mm. in diameter and appear upon the cut surface as grayish pin point bodies. Histologically they resemble the structure of a lymph node. The central or germinal area of lymphocytes contains numerous mitotic figures in early

life and the peripheral zone is sharply differentiated from the surrounding tissue. Thus, the two essential anatomic components consist of (1) The reticulo-endothelial system, composed of the pulp cells and their supporting reticulum, and (2) the lymphoid follicles or Malpighian bodies. The latter are abundant and active in early life but undergo progressive atrophy with advancing years.

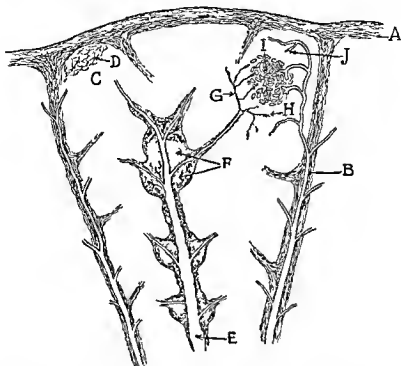


FIG. 181.—Splenic lobule (modified after Jordan). *A* Tunica albuginea covered by serosa. *B* trabecula containing vein. *C* splenic unit. *D* reticulum. *E* artery of lobule. *F* Malpighian bodies with germinal center. *G* penicilli. *H* ellipsoid. *I* splenic pulp. *J* venous sinuses.

### PHYSIOLOGY OF THE SPLEEN

The spleen is the largest member of two important systems, the lymphoid and the reticulo-endothelial, and its circulatory and certain functional activities are closely interrelated with those of the liver. The lymphoid system, sharing in the production of lymphocytes, is especially active in childhood and apparently plays an immunity rôle in certain chronic infections, especially tuberculosis.

The reticulo-endothelial activities are various. (1) Erythrocytes are chiefly prepared for ultimate destruction, in this process some bilirubin is formed and the iron content is conserved for further hemoglobin formation. (2) Bacteria and particulate matter circulating in the blood are removed by the filter-like structure and

phagocytic process of the pulp cells (3) Antibodies are formed (4) Some substance or substances are produced which exert a regulatory influence upon bone-marrow in not only controlling red cell production but also in affecting the stability of the cells as regards hemolysis (5) The coagulation time of the blood is influenced

The splenic capsule contracts about once per minute. The rhythmic contraction and expansion of the pulp alter the blood content of the organ and may account for the rarity of neoplastic metastases. Approximately 20 per cent of the portal blood entering the liver comes from the splenic reservoir. The greatly increased volume in splenomegaly may definitely alter hepatic function. Whether the diseased spleen also contributes toxic substances is questionable.

**Removal of the Normal Spleen.** This procedure results in (1) Polymorphonuclear leukocytosis followed by relative lymphocytosis (2) temporary anemia with reduction in both erythrocytes and hemoglobin (3) hyperplasia of other elements of the reticulo-endothelial system and (4) possible increased susceptibility to infection.

**Removal of the Diseased Spleen.**—In certain instances this is followed by (1) An increase in both erythrocytes and hemoglobin due to the preservation of defective or immature red cells previously destroyed by the spleen (2) reduction in the portal blood stream volume. In splenomegaly a large percentage of the portal blood may be splenic and the ascites in portal cirrhosis may disappear following splenectomy (3) relief from hepatic toxemia through the removal of toxic substances metabolized in the diseased spleen (4) the relief of jaundice when due to excessive splenic destruction of the erythrocytes and (5) reduction in the clotting time. The diseased spleen may not only destroy red blood cells but also the platelets and thereby prolong the clotting time with a tendency to hemorrhage.

### CONGENITAL ABNORMALITIES OF THE SPLEEN

Congenital absence of the organ is very rare and micro splenia is noted uncommonly. Neither condition produces impairment of health. Accessory splenic masses varying from 1 to 3 cm. in size occur not infrequently in the region of the hilus and occasionally in the great omentum. A wandering or ptosed spleen may result from congenital or acquired elongation of the peritoneal folds. In the rare cases of visceral transposition the spleen lies beneath the right dome of the diaphragm.

### CYSTS OF THE SPLEEN

Cysts of the spleen are rare. They may be classified as non-parasitic and parasitic.



**Non parasitic Cysts** — These comprise the following (1) Solitary cysts (2) multiple cysts and (3) polycystic degeneration (1) The solitary cysts are of chief surgical interest. They are usually unilocular and may attain considerable size. The cyst wall is composed of connective tissue and the contents generally consist of old blood at times however the fluid is clear serum. The majority result from a previous hematoma and are often termed hemorrhagic cysts. (2) Small multiple cysts are not unusual. They rarely produce symptoms and have no clinical significance. (3) Polycystic degeneration occurs but rarely. The numerous cysts may fuse and thus replace a portion of the spleen or its entirety.

**Treatment** — Only large or inflamed cysts which produce symptoms require treatment. Splenectomy is best performed as enucleation is seldom feasible.

**Parasitic Cysts** — The spleen is involved in approximately 2 per cent of echinococcus infections and in one-half of the cases the disease is confined solely to the organ. The cysts usually develop in the central portion and at times attain large size. Associated perisplenitis may produce firm adhesions to surrounding structures especially the diaphragm. Unless removed the cysts may rupture and contaminate the peritoneal cavity or develop suppuration.

The growing cyst may remain asymptomatic for months until pressure or adhesions produce local discomfort. A large non tender mass in the splenic region associated with eosinophilia should at once suggest the diagnosis.

The prognosis is favorable if the disease is limited to the spleen and the organ is removed intact. In the presence of dissemination or suppuration the outcome is grave. Splenectomy should be performed early before adhesions develop and prevent removal. When this is impractical the cyst should be incised and the cavity marsupialized.

### TUMORS OF THE SPLEEN

Benign tumors are pathologic curiosities and comprise angioma, fibroma, chondroma, osteoma, myoma and myxoma. They rarely produce symptoms and are discovered accidentally. Treatment comprises splenectomy.

Although malignant tumors are extremely rare the splenic lymphoid structure is subject to the same type of neoplasms as occur in the lymph nodes such as fibrosarcoma, lymphosarcoma and endothelioma or large cell sarcoma. Malignant angiomas have also been reported.

The first symptom to attract attention is either the presence of a mass or a dull dragging ache in the left hypochondrium. In the absence of metastasis splenectomy should be performed. In inoperable cases irradiation is often of palliative value.

## INJURIES OF THE SPLEEN

The normal spleen is highly vascular and relatively soft and brittle. In diseased conditions these elements are exaggerated. The organ is subject to contusion, laceration and rupture either from subcutaneous injuries due to blows, kicks and especially run over crushing accidents or puncture wounds resulting from rib fracture, shooting or stabbing. In rare instances spontaneous rupture may occur in the splenomegaly of malaria, typhoid or other diseases.

**Pathology**—Subcutaneous injuries may occur with or without rupture of the splenic capsule. In the latter condition termed contusion of the spleen the parenchymal laceration produces intra capsular hemorrhage. The resulting hematoma may eventuate in resolution, cyst formation or rarely suppuration. Trauma of greater severity may cause rupture of the spleen through laceration of the capsule. The actual splenic damage may vary from that of a slight fissure to pulpification. The free extravasation of blood into the peritoneal cavity is generally profuse and with rupture of the main splenic vessels death may occur within a few minutes. Associated lesions of the liver, pancreas, kidneys or hollow viscera may accompany the splenic injury.

**Symptomatology**—Mild contusions may escape notice. More severe damage produces local pain, tenderness, muscular rigidity and an increase in size of the spleen. Not infrequently the pain is referred to the left shoulder. In splenic rupture the symptoms of intra-abdominal hemorrhage and shock vary in proportion to the vascular damage. The general symptoms of restlessness, thirst, pallor, increasing pulse rate, leukocytosis and polymorphonuclear leukocytes are accompanied by local tenderness and diffuse abdominal rigidity. Dulness in the left flank may also be exhibited.

**Prognosis**—*Splenic rupture is lethal in over 80 per cent of the cases treated conservatively.* Early operation with suitable supportive measures salvages approximately 70 per cent.

**Treatment**—Symptoms of internal hemorrhage demand immediate exploration. Although a precise anatomic diagnosis is not always possible, it is well to remember that the site and type of injury frequently indicate the probable pathology. Blood transfusion just before or during the operation is often life-saving when impractical. 10 per cent glucose in physiologic saline solution should be administered by infusion.

Splenic rupture is best treated by splenectomy as suture of the friable pulp is impractical. Occasionally an adherent spleen will necessitate tamponade and in rare instances a clamp may be applied to the pedicle and splenectomy performed forty-eight hours later. Following operation the patient should be placed in bed in the

shock position and covered with warm blankets surrounded by hot water bottles. Abundant saline solution should be administered by proctoclysis, hypodermoclysis or phlebotomy. Cases which survive the first twenty-four hours usually recover. Subsequent transfusion is indicated if the hemoglobin is below 40 per cent.

### ABSCESS OF THE SPLEEN

Splenic abscess occurs rarely and is seldom diagnosed. Most cases are due to metastatic invasion and the primary disease usually typhoid fever or sepsis overshadows the secondary. At times an abscess develops from hematogenous infection of a traumatic intracapsular hematoma and occasionally without apparent cause.

**Pathology**—Practically all the pyogenic bacteria except the gonococcus have been recovered in splenic abscess. Whereas metastatic lesions are usually multiple those of traumatic or septic infarctal origin are generally single. A portion of the pulp or the entire organ may become necrotic and the capsule may ultimately rupture and produce a subdiaphragmatic abscess.

**Symptomatology** Local symptoms are generally wanting until perisplenitis develops when the enlarged spleen becomes painful and tender. Fluid occasionally accumulates in the left pleural cavity and fixation of the left dome of the diaphragm may be evidenced upon fluoroscopic examination. High leukocytosis is common.

**Diagnosis**—The diagnosis is difficult and the pathology may be confused with empyema, perinephritic or subphrenic abscess. Exploratory aspiration is a helpful diagnostic aid.

**Treatment**—When the abscess is intracapsular splenectomy is indicated providing it may be performed without contamination of the peritoneal cavity. Large suppurative accumulations require incision and drainage; the approach may be abdominal, transpleural or retroperitoneal.

### TUBERCULOSIS OF THE SPLEEN

Although the actual occurrence of primary tuberculosis of the spleen is problematic a few cases have been described in which an apparent solitary focus occurred in the organ. The chief symptoms are pain and splenic enlargement associated with loss of weight and strength and slight pyrexia. The red cell count is usually increased. Splenectomy in such cases is justifiable and may result in cure.

Secondary tuberculosis of the spleen is a common occurrence in children who succumb to the disease. It should be noted that calcified tubercles may produce opacities in splenic roentgenograms.

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Secondary tuberculosis of the spleen is a common occurrence in children who succumb to the disease. It should be noted that calcified tubercles may produce opacities in splenic roentgenograms.

### SYPHILIS OF THE SPLEEN

The spleen is frequently involved in congenital hereditary and acquired lues. The question of splenectomy rarely arises except in cases of tertiary syphilis in which the enlarged organ may harbor and protect the spirochetes from the action of arsphenamine.

### SPLENIC ANEMIA

#### (INCLUDING BANTI'S DISEASE)

Poole and Stillman define splenic anemia as a disease of unknown etiology and chronic course characterized by an enlargement of the spleen which is often enormous and which shows an interstitial splenitis, a secondary anemia of moderate severity, a marked tendency to hemorrhage from the mucous membranes and into the skin, and in the terminal stages cirrhosis of the liver and ascites.

This simple yet comprehensive classification is of definite clinical value. The following diseases are excluded: leukemia, von Jaksch's anemia, pernicious anemia, hemolytic icterus, Gaucher's disease, purpura hæmorrhagica, malaria and syphilis. A rather large group of unsatisfactorily differentiated anemias are included, especially Banti's syndrome. The latter is reserved for those cases in which ascites accompanies the pathology. Accordingly, Banti's syndrome (disease?) may be the terminal stage of many cases of splenic anemia.

**Etiology**—The disease is more common in females and affects both children and young adults. Syphilis may be an associated factor. The cirrhosis of the liver is apparently a secondary manifestation and may result from the same toxin or toxins which produce the splenic anemia. Von Jaksch's anemia is considered by some authorities to be a special type of splenic anemia occurring in early life.

**Pathology**—At times the spleen is enormous. The associated perisplenitis commonly produces firm anchorage to adjacent organs, especially the diaphragm; also the splenic veins may be sufficiently fibrotic to render ligation difficult. The cut surface of the organ is firm and dry, the trabecule conspicuous, and the follicles reduced in size and number.

Histologic examination reveals a diffuse interstitial splenitis with eccentric follicular fibrosis. Strands of fibrous tissue appear as extensions from the thickened trabecule; the pulp cells are diminished in number, and the lymphoid follicles are atrophic or more often absent. The portal veins may show evidence of fibrosis and the liver cells exhibit a definite cirrhosis. The ascites which appears later may be accompanied by varicosities of the lower portion of the esophagus.

**Symptomatology**—The onset is insidious dragging pain from splenic weight is frequently the earliest symptom or progressive pallor may first attract attention. The anemia is of secondary type and often exhibits remissions and exacerbations. The erythrocytes may be reduced by one-half and the color index to 0.7 or 0.5 erythroblasts are not uncommon. The leukocytes are reduced in number and evidence a decrease in polymorphonuclear neutrophils and an increase in large mononuclears. The bleeding and coagulation times remain normal.

A hemorrhagic tendency may appear at any time the bleeding being more pronounced in cases having a low platelet count (thrombocytopenia). Although the hemorrhages into the skin and mucous membranes are seldom serious hematemesis may at times prove fatal. Clinical jaundice is infrequent but latent jaundice is commonly demonstrable.

The foregoing symptoms accompanied by progressive splenic enlargement represent the primary stage of the disease and may continue for several years. The secondary stage is associated with definite liver changes and lasts from six to eighteen months. The liver becomes enlarged and the hard non-tender border may extend far below the costal margin. Finally the terminal stage of portal cirrhosis is evidenced by the development of ascites (Banti's syndrome). Death usually occurs within two years thereafter from anemia, hemorrhage or intercurrent infection especially bronchopneumonia. Although the disease pursues a slow chronic course it is ultimately fatal unless checked by splenectomy.

**Diagnosis**—This is made chiefly by the process of exclusion. When splenomegaly develops without apparent cause and the condition is associated with anemia and leukopenia the diagnosis of splenic anemia is justifiable. In *hemolytic jaundice* icterus is constantly present the erythrocytes exhibit increased fragility the urine contains urobilin and a familial history commonly obtains. *Pernicious anemia* presents a characteristic blood picture accompanied by remissions and exacerbations. In *Gaucher's disease* the conjunctival lesions, skin pigmentation and familial tendency are helpful diagnostic aids the spleen is generally larger the anemia less pronounced and ascites is rare. In *portal cirrhosis* splenic enlargement is secondary and the anemia is less marked. In *Hansen's cirrhosis* the liver is greatly enlarged and accompanied by jaundice. *Luetic* enlargement of the liver and spleen exhibits positive serologic findings and other evidences of the disease. In *Jakob's anemia* is associated with leukocytosis and at times enlargement of the superficial lymphoids.

**Treatment**—Early splenectomy is the only known cure for splenic anemia. In some instances however there is a residual tendency to hemorrhage even years after operation. This diathesis

is chiefly evidenced in the thrombocythemic types apparently they are less subject to influence by splenectomy than the thrombocytopenic group. Late splenectomy in the presence of advanced cirrhosis is only palliative. In suitable cases fortified by transfusion the mortality of splenectomy is less than 10 per cent.

### VON JAKSCH'S ANEMIA

Von Jaksch's anemia is a disease of early life characterized by enlargement of the liver and spleen and at times of the superficial lymph nodes. The blood exhibits marked anemia and a persistent though variable leukocytosis. The latter is sometimes sufficient to be termed pseudoleukemia. Nucleated red blood cells occur frequently at times in large numbers.

**Etiology**—The cause is unknown the reaction of the infantile blood-forming elements may be due to any of a number of toxins. Rickets, syphilis, tuberculosis and gastro-intestinal disturbances are commonly associated conditions. The disease is best considered as a clinical rather than pathologic entity.

**Pathology**—The most striking feature is the enlarged hard spleen which fails to exhibit any characteristic changes. There is generalized fibrosis and the follicles normally abundant in early life are small or absent. The pulp is generally hyperplastic. The liver is enlarged and evidences areas of hematopoiesis. The sectioned lymph nodes are at times cherry red and the bone-marrow may exhibit hyperplasia.

**Symptomatology**—The dominant symptoms of splenic enlargement and anemia develop insidiously chiefly during the first two or three years of life but seldom prior to six months. The splenomegaly develops early in the disease and may be extreme the lower edge of the spleen even extending to the iliac crest. Enlargement of the liver occurs late and is not pronounced. Although the skin may exhibit an icteric tint true jaundice does not occur. The superficial lymph nodes generally become palpable.

The blood picture exhibits progressive anemia which at times is severe. The marked fall in both erythrocytes and hemoglobin is usually accompanied by a low color index at times however this may be high and the frequent occurrence of normoblasts and megaloblasts may suggest pernicious anemia. The fragility of the red cells is normal. Leukocytosis is constant varying from 10,000 to 50,000 the degree however bears no relationship to the severity of the disease. The lymphocytes may be relatively increased and myelocytes are commonly present in large numbers. The urine frequently contains urobilin and rarely bilirubin.

**Diagnosis**—The dominant symptoms are anemia, splenomegaly and leukocytosis. *Myelocytic leukemia* is differentiated by the



greater proportion of leukocytes and myelocytes and more rapid fatal termination and *lymphocytic leukemia* by the great relative and absolute lymphocytosis. *Pernicious anemia* rarely occurring in early life exhibits a high color index and gastric achylia and leukocytosis is absent. *Splenic anemia* evidences leukopenia and a tendency to hemorrhages. In *hemolytic jaundice* there is persistent icterus and increased erythrocytic fragility.

**Course and Prognosis** — The course of the disease is chronic and the prognosis is uncertain. Approximately 25 per cent of the cases die within a year from bronchopneumonia, diarrhea or other complications. The tendency to spontaneous recovery is most favorable in the presence of some underlying condition which is amenable to treatment such as rickets or syphilis.

**Treatment** — This comprises the treatment of any associated disease such as rickets, tuberculosis or syphilis, hygienic dietetic measures including heliotherapy, hematinics and repeated supportive transfusions.

Splenectomy is only advisable in progressive cases which fail to respond to other remedial measures. The therapy is based upon the hypothesis that a pathologically enlarged spleen is a destroyer of relatively sound red blood cells. Cases appear to be uniformly improved after operation even though the blood picture may never return to normal. An erythroblastic crisis frequently follows and the nucleated red blood cells may outnumber the leukocytes for an indefinite period. A variable degree of anemia generally persists. The marked general improvement and occasional complete cures fully warrant splenectomy in suitable cases.

### CHRONIC HEMOLYTIC ICTERUS

The disease is characterized by splenomegaly, jaundice, secondary anemia, increased fragility of the erythrocytes and the absence of acholic stools and bilirubinuria. Two types are observed, the congenital and familial, termed the Chauffard-Minkowsky form, and the acquired or Hayem-Widal variety. Both are described in the chapter on Icterus.

The congenital and familial type concerns the pediatric surgeon. Whether the disease is essentially an increased fragility of the erythrocytes due to dysfunction of the bone-marrow or is primarily of splenic origin remains controversial. Judging from the results of splenectomy, either may be the dominant factor. In some instances the reestablishment of normal resistance of the red blood cells suggests perversion of splenic function; in others the erythrocytic fragility persists although the patient is apparently cured.

**Pathology** — The spleen is usually only moderately enlarged. There is mild generalized fibrosis and the thickened capsule may be

accompanied by perisplenitis. The pulp is congested and frequently exhibits active erythrocytic phagocytosis; the follicles are reduced both in size and number. The enlarged liver may show biliary cirrhosis. Erythroblastic activity is evidenced in the bone-marrow and increased pigmentation occurs in the spleen, liver, bone-marrow and kidneys.

**Symptomatology.** The jaundice may be present at birth or develop in early infancy. When once developed it persists although the degree is strikingly variable. The patients are subject to acute attacks of hemolysis which are accompanied by pain, pyrexia and exacerbation of the icterus. Between attacks the jaundice may be extremely slight and the patient feel well. The red blood cells persistently exhibit a diminished resistance to hemolysis. The secondary anemia varies in severity and nucleated red blood cells appear in considerable numbers after each crisis. The leukocytes remain normal except for a slight increase during the acute attacks. The urine often contains a large amount of urobilin; bilirubinuria is absent.

**Treatment.** Many cases appear more jaundiced than sick and require no treatment. Splenectomy is rarely indicated during childhood and only when the disease is rapidly progressive and fails to respond to remedial measures including repeated blood transfusions. Operation is generally followed by permanent symptomatic cure despite the fact that in many instances the erythrocytic fragility persists.

### GAUCHER'S DISEASE

In this rare form of splenomegaly the increased size of the organ is due to the growth of certain large cells of rather definite morphology which are believed to be derived from the reticulo-endothelial system. The etiology is unknown. The condition is often familial but not hereditary and females are more frequently affected.

**Pathology.**—The huge spleen is crowded with characteristic masses of large multinuclear cells of endothelial character (Gaucher's cells); similar cells occur in the liver, bone-marrow, lymphatic tissue and suprarenals. The disease appears in infancy or early childhood and the condition is probably congenital. (Fig. 182.)

**Symptomatology.**—The chief symptom is progressive enlargement of the spleen. The hard, smooth, non-tender organ may reach enormous size. After a time the liver enlarges but ascites is rare. Although the general health may not be affected for months or years anemia of the chlorotic type ultimately develops. Leukopenia is quite characteristic; the differential count is preserved but the platelets may be diminished. In over one-half the cases a brownish pigmentation occurs on the parts of the body exposed to light; less frequently a wedge-shaped thickening of the con-

junctiva develops on each side of the corner. The superficial lymph nodes often become palpable and subcutaneous or submucous hemorrhages may occur. Persistent bone pain may be followed by pathologic fracture. At times mental deficiency is evidenced. The disease is slowly progressive and the average duration exceeds twenty years. Death may occur from intercurrent infection.

**Diagnosis** — The diagnosis is impossible in the early stages. Splenic puncture has been recommended but is not advisable. The late discoloration of the skin and conjunctival changes are characteristic differentials.

**Treatment** — Any form of treatment is necessarily symptomatic as the disease affects the entire hematopoietic system. Splenectomy has been performed many times; it may not only afford relief from the splenic drag but also bring about general improvement.

### PURPURA HÆMORRHAGICA

Idiopathic purpura hæmorrhagica (*thrombopenia Werlhof's disease*) is a disease of early life which chiefly affects children between the ages of three and ten years. The dominant symptom is hemorrhage. Spontaneous bleeding may occur from any of the mucous membranes, nose, gums, gastro-intestinal tract or endometrium, into the skin in the form of petechiæ or ecchymoses and at times into the joints.

**Etiology** — The cause is unknown and the pathogenesis is obscure. Heredity is an occasional influence. The blood platelets are definitely diminished, the normal number of 400,000 per cubic millimeter being commonly reduced to 100,000 or less. Following splenectomy the platelets may promptly return to normal or remain unaltered. Since in either instance the tendency to hemorrhage is permanently checked, the spleen is apparently responsible for the bleeding. Rosenthal suggests there may be two types of purpura hæmor-



FIG. 189. Gauche's disease.

rhagica in one the essential disturbance is splenic and the platelets return to normal following removal of the organ in the other the dominant element is actual platelet disturbance and the numerical deficiency persists

**Pathology** —The spleen is enlarged in about one-third of the cases but characteristic lesions are wanting In some instances the organ appears normal whereas in others it is packed with platelets undergoing phagocytosis The bone-marrow remains unaltered The bleeding time is prolonged from several minutes to even an hour or longer The red blood cells and hemoglobin are only diminished after severe hemorrhages The leukocytes may remain normal or exhibit slight leukocytosis or leukopenia

**Symptomatology** —Although vague prodromal malaise may occur the onset is generally abrupt Profuse nasal hemorrhage is most common also bleeding from the gums hematemesis or hematuria occur less frequently and hemoptysis rarely Ecchymoses may appear upon the visible mucous membranes and large cutaneous areas may become involved Hemarthrosis may also occur although less often than in rheumatic purpura In severe attacks nephritis may develop Rarely the loss of blood is sufficient to produce collapse

**Diagnosis** The salient differential characteristic is the marked reduction in the number of platelets they are frequently less than 70 000 and at times none is demonstrable Coagulation is relatively normal but the bleeding time may be so greatly increased that even such a procedure as skin puncture is actually dangerous During the active stage of bleeding the application of a tourniquet about an extremity for five minutes will cause petechiae to appear below the constriction (Refer to Chapter VII)

**Course and Prognosis** —The duration of the disease in favorable instances averages about two weeks Recurrence however are common and repeated attacks may render the patient a chronic invalid The prognosis in general is good and most cases recover Fatalities are usually due to severe repeated bleeding cerebral hemorrhage or intercurrent infection

**Treatment** Mild cases generally respond to single or repeated transfusions Citrated blood however is contraindicated In 1916 Haznelson performed the first splenectomy for hemorrhagic purpura with striking and immediate benefit Many cases have since been reported It is now generally recognized that *splenectomy is more effective in essential thrombopenia than in any other condition* Even before the wound is closed the bleeding time which may have been prolonged to an hour or longer is often reduced to less than ten minutes The platelets rapidly increase to normal or above Despite the fact that in some instances there is a subsequent reduction of the platelets to the preoperative count further hemorrhage

does not occur. This definite improvement obtains in 75 to 80 per cent of splenectomized patients and the cures appear to be permanent. Failures have been attributed to the presence of accessory splenic tissue. In a few instances splenic radiation has restored the platelet count to normal.

### MISCELLANEOUS DISEASES OF THE SPLEEN

**Cirrhosis of the Liver**—Portal and biliary cirrhosis are rare in early life. In the portal type with ascites, and possibly also in Hanot's cirrhosis, splenectomy may be helpful in cases of marked splenomegaly. In addition to diminishing the portal blood volume, certain toxins of splenic origin may also be removed.

**Tropical Splenomegaly**—Splenic enlargements occurring with malaria, kala-azar, relapsing fever, and Egyptian and Columbian spleen are all included under this general term. The diseases are seldom seen in the United States. In cases of marked splenomegaly, removal of the organ may rarely be indicated for symptomatic pressure relief.

### INDICATIONS FOR SPLENECTOMY

**Traumatic Lesions**—Severe splenic laceration and rupture are preferably treated by splenectomy. Suture of the friable pulp is unsatisfactory.

**Cysts**—Small multiple cysts are inconsequential. Large cysts require splenectomy, although cases complicated by dense adhesions are more safely treated by incision and drainage; a chronic sinus usually follows such procedure. Polycystic degeneration also calls for removal of the organ. Echinococcus cysts should be removed *en masse* by splenectomy, aspiration should be avoided.

**Abscess**—Removal of the organ should always be performed unless there is danger of peritoneal contamination through rupture, in the latter case, and in the presence of dense perisplenitis drainage is indicated.

**Tumors**—Splenectomy should always be performed, enucleation of the tumor is dangerous as a positive diagnosis of benignancy is often impossible.

**Splenic Anemia Including Banti's Disease**—Early splenectomy produces marked improvement or cure, and in the later stages, improvement and prolongation of expectancy.

**Hemolytic Jaundice**—Splenectomy is indicated in progressive cases and generally results in symptomatic cure.

**Purpura Hæmorrhagica**—Except in mild cases, splenectomy should be performed promptly, the results are immediate, striking and permanent.

*Iron Deficiency Anemia* — Splenectomy is indicated when remedial measures including irradiation and repeated transfusions are without benefit. Removal of the organ is followed by cure or definite improvement.

*Gaucher's Disease* — Splenectomy is only recommended for mechanical relief. Improvement in the anemia may follow.

*Tuberculosis* — In primary tuberculosis of the spleen removal of the organ is definitely indicated, in secondary involvement operation is interdicted.

*Leukemia* — In chronic myelogenous leukemia following reduction in the size of the organ by irradiation, splenectomy may prolong life but is never curative.

*Cirrhosis of the Liver* — Splenectomy is rarely indicated in childhood.

*Malaria and Tropical Splenomegaly* — In exceptional cases splenectomy may become advisable for symptomatic pressure relief.

**Contraindications** — Splenectomy is contraindicated in Hodgkin's disease, lymphatic leukemia, uncomplicated rickets, gastric hemorrhage, syphilis and actinomycosis.

**Irradiation** — In the various anemias associated with splenomegaly splenectomy should never be advocated except after well-considered study. Remedial measures including repeated transfusions and especially irradiation should always be given a trial. Roentgen ray therapy, when competently administered is often of great benefit. The splenomegaly may not only be reduced in size but marked constitutional improvement often occurs. With proper dosage the danger of producing perisplenitis appears more theoretical than real. Radium packs however are not advised. In many instances preliminary irradiation will minimize the danger of subsequent splenectomy.

## SPLENECTOMY

Splenectomy is a relatively simple procedure except when the organ is friable or firmly bound to surrounding structures by perisplenitis. Before attempting removal the position of the tail of the pancreas in relationship to the hilus should always be ascertained. General anesthesia is advisable.

**In Traumatic Rupture Cases** — A 4 inch upper left split rectus incision is commonly employed. The organ is delivered into the wound and after noting the position of the tail of the pancreas the pedicle is transfixed and ligated with a double ligature of No. 2 plain catgut. The pedicle is then divided and the spleen removed. (Fig. 183.)

After the free blood in the peritoneal cavity has been aspirated the surrounding structures including the liver should be carefully inspected for concomitant lesions. The peritoneum and posterior rectus sheath are approximated with No. 2 plain catgut suture and

the anterior sheath with No 2 chromic. Retention sutures of dermal or silkworm are advisable.

The importance of supportive blood transfusion or the intravenous injection of 10 per cent glucose in physiologic saline either before or during operation is discussed in the section on Injuries of the Spleen. During the first twenty four hours following operation the foot of the patient's bed is elevated on 8 inch blocks and the patient is kept warm with blankets surrounded by hot water bottles. The water saline balance should be adequately maintained.

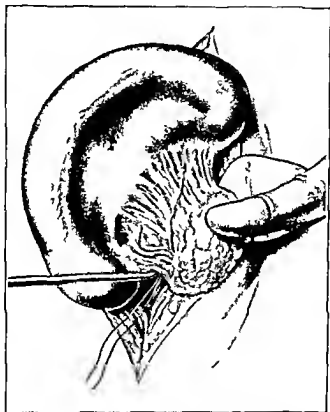


FIG. 183.—Ligature of the splenic pedicle by transfixion clasp. The tail of the pancreas is carefully retracted.

through the aid of infusions hypodermoclyses or proctoclyses of glucose in normal salt solution. Subsequent transfusion is indicated if the hemoglobin remains below 40 per cent. Cases surviving the first twenty four hours usually recover.

**In Cases of Large Splenomegaly**—Various incisions have been devised to afford ample exposure. A left split rectus incision may be curved across the muscle at its upper extremity to the mid line (Bevan) or the upper angle of the incision may be extended outward across the rectus and include the obliquus and transversalis muscles.

After adequate exposure is obtained the spleen is explored for adhesions especially to the diaphragm. To prevent serious hemorrhage in cases complicated by extreme perisplenitis preliminary ligation of the pedicle is sometimes advantageous. This may be performed as follows: the anterior edge of the spleen is rotated to the left and the gastrosplenic omentum is divided thus exposing the pedicle. The splenic vessels are then ligated with care to prevent the inclusion of pancreatic tissue.

Adhesions in the average case may be separated by passing the hand between the spleen and diaphragm. Dense bands are divided between ligatures and oozing is controlled by hot pads. After ligation and division of the gastrosplenic omentum the spleen is delivered by withdrawing the lower pole first. The organ is then turned to the right and the tail of the pancreas is identified and bluntly separated from the hilus. The large and often friable vessels of the pedicle are ligated by a series of double No. 2 plain catgut chain ligatures passed on carriers (Fig 183). Following removal of the spleen the pedicle is reinspected for bleeding. The pads are then withdrawn from the splenic bed and any oozing points are ligated or coagulated with the endotherm current. The wound is closed in layers without drainage.

The mortality from splenectomy is chiefly due to hemorrhage shock and postoperative pneumonia. Adequate preoperative preparation of the patient including oral and nasopharyngeal prophylaxis is highly important. Shock may be minimized by the transfusion of blood during operation. The gentle separation of bands and adhesions and meticulous hemostasis.

Autohemotransfusion appears to reduce the incidence of postoperative pulmonary complications. After closure of the wound 5 to 10 cc. of the patient's blood is withdrawn from a vein and immediately reinjected into the gluteal muscles. The method is without danger.

## THE PANCREAS

Surgical diseases of the pancreas are very rare in children and the pediatric surgeon is concerned chiefly with injuries to the organ especially those resulting from crushing in run-over accidents.

**Congenital Abnormalities**—Opie found an accessory pancreas 10 times in 1800 autopsies. The supernumerary tissue occurs chiefly in the musculature of the stomach and upper intestine seldom exceeds the size of a pea and is inconsequential. In some instances accessory pancreatic tissue may be present in hypertrophic pyloric stenosis. Hale considers it a dominant factor.

Being a fixed retroperitoneal organ the pancreas is seldom displaced. In rare instances it may comprise one of the visceral elements in large congenital exomphalos.



**Pancreatitis** — Acute subacute or chronic inflammation of the gland rarely occurs in early life

**Cysts and Tumors** — Cystadenoma and lymphosarcoma are exceedingly rare Retention degenerative and pseudocysts are usually the residuum of pancreatitis in adult life in childhood they may rarely follow trauma Benign tumors fibroma and lipoma are pathologic curiosities

**Injuries** — The depth of the pancreas affords it comparative protection Injury may occur directly from a stab or bullet wound or indirectly from crushing in run over accidents also from kicks in the abdomen or falls from great heights the dorso lumbar spine acting as a point of counter pressure

**Pathology** — Slight contusion of the pancreas may produce pancreatic concussion (miliary hemorrhage) Recovery follows and the condition may eventuate in fibrosis (chronic pancreatitis) Although a greater trauma may produce damage within immediate safe limits subsequent leakage of pancreatic juice often occurs Severe injuries may cause partial tears with resulting hemorrhage and necrosis Whereas leakage into the lesser sac may be walled off at the foramen of Winslow by fibrinous exudate and produce a pseudocyst the escape of pancreatic juice into the free peritoneal cavity produces chemical peritonitis Fat necrosis is evidenced in eight to ten hours and the peritonitis is often complicated by bacterial invasion Unless the fluid is evacuated and drained the process becomes rapidly lethal In severe tears involving the splenic vein death may occur from hemorrhage within a few minutes

**Symptomatology** — The immediate symptoms of severe pancreatic damage are those of shock and after a day or two of apparent improvement acute fatal peritonitis develops The early symptoms of severe epigastric pain tenderness and rigidity demand immediate exploration (Refer to Intra abdominal Injuries) Hemorrhage from neighboring organs should not distract attention from the pancreas and in all serious lesions of the stomach spleen or gastroduodenal vessels the lesser omentum should be opened to examine the organ

**Treatment** Damage of the pancreas is best treated by suture of its surface when possible or by tamponage In injuries to the tail of the organ amputation is preferable Drainage in all cases is imperative Since a pancreatic fistula may discharge for weeks the wound area should be covered with Lassar's paste to prevent digestion of the abdominal walls

Stab or bullet wounds to the epigastrium generally involve the stomach or stomach and transverse colon In such injuries the pancreas should always be examined as above described Early drainage is often life-saving even following a tear of the main excretory duct

## CHAPTER XXXIV

### THE COLON

#### DEVELOPMENT OF THE COLON

FIG 184 is the schema of the human embryonic intestinal canal with the umbilical loop and mesenteric attachments at about six weeks. During the third month the portion of the large intestine

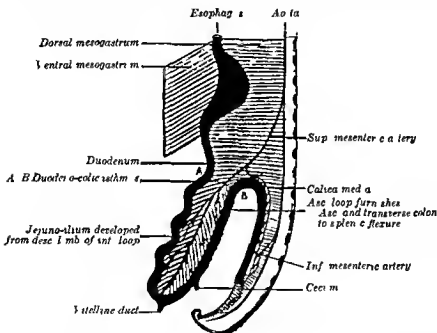


FIG 184 Schema of the embryonic intestinal tract at six weeks (Brenner courtesy of the Am Jour Surg)

developed from the ascending limb rotates to the mid line and contacts the ventral abdominal wall. It then passes ventrad of the jejunum-ileal coils toward the cephalic end of the abdominal cavity and lies transversely along the greater curvature of the stomach. The rapidly growing coils of small gut developing from the descending arm of the loop crowd the colon more and more cephalad. In the fourth month the cecum turns to the right, comes into contact with the under surface of the liver and subsequently reaches the ventral surface of the right kidney. Its further descent to the right

iliac fossa is complete at birth, or soon thereafter. High cecal situs after the first year denotes premature arrest from faulty descent.

With full development, the cecum is normally invested with peritoneum and may have a short mesentery; the ascending colon is incompletely covered by peritoneum, being adherent to the posterior parietes; the transverse colon is loosely swung across the abdomen in a broad mesenteric hammock, being firmly anchored at the hepatic and splenic flexures, the latter at a considerably higher angle than the hepatic; the descending colon is supplied with a very short fixation mesentery, and the sigmoid with a pendulous mesentery.

### MALFORMATIONS OF THE COLON.

In rare instances the cecum is absent and the ileum enters the colon directly; or the ascending colon may be wanting and the ileum joins the transverse colon. A long cecal mesentery is a common malformation. The resulting "cecum mobile" may descend into the pelvis and predispose to stasis and volvulus. Occasionally an elongated common mesentery is supplied to the terminal ileum, cecum and ascending colon, and very rarely the entire small and large intestine retain their single embryonal type of mesentery. (Refer to Total Volvulus.)

**Transverse Colon.**—The transverse colon is freely movable and in visceroptosis may lie partially in the pelvis. Its length is subject to considerable variations and it is often elongated; in rare cases it is almost entirely absent and the ileum enters at the splenic flexure.

**Sigmoid.**—Unusual elongation is relatively common. In a small number of cases the sigmoid opens into the rectum on the right side, and in visceral transposition the bowel is located on the right side.

### CONGENITAL ABNORMALITIES OF THE COLON.

These are of rare occurrence and comprise the following: failure of rotation; absence of the colon; double-barrel colon; microcolon; and megacolon.

**Failure of Rotation**—In the rare anomaly of visceral transposition the colon lies in the reverse of its usual location and the appendix occupies the left lower quadrant. Incomplete rotation occurs more frequently and the cecum becomes arrested at some point of its normal circuit, most commonly under the liver or in the left upper quadrant. Whereas the cecum may normally occupy a rather high position at birth, such situs after the first year denotes premature arrest from faulty descent. Rotation abnormalities are readily demonstrable roentgenologically.

**Total Absence of the Colon** — This abnormality is very rare. More often the missing segment is replaced by a fibrous cord. An umbilical fecal fistula may accompany the condition.

**Double-barrel Colon** — The condition is exceedingly rare. It may occur in either the ascending or descending colon, the bowel presenting two lumina separated by a fibrous septum. One or both barrels may functionate.

**Microcolon** — This is an extremely rare abnormality and but few cases have been observed. The condition may represent a segmental atresia or the entire bowel may be involved. Extreme types produce obstruction in the first week of life.

### CYSTS OF THE COLON

Cysts of the large bowel are rare and comprise the following: **inclusion cysts**, resulting from inflammatory closure of the ostia of the intestinal glands, **degeneration cysts**, associated with sarcoma and **polypoid coli** and **parasitic cysts**. The last occur chiefly in the mesentery rather than in the bowel wall.

**Embryonal enterocysts** may invade the colon secondarily. Arising from fetal remnants they develop mainly in the terminal ileum. The cysts are multiple and often attain large size. Not infrequently they extend through the ileocecal valve into the colon and produce symptoms of obstruction. Infants are chiefly affected. Ileocecal resection may be required for their removal.

### TUMORS OF THE COLON

**Benign Tumors** — Benign growths of the colon are of rare occurrence in early life. They comprise fibroma, lipoma, angioma, myoma and myxoma. In lymphatic leukemia and Hodgkin's disease masses of lymphoid tissue may develop in the bowel which simulate tumor formation.

Angiomas, especially of the cavernous type, may produce hemorrhage into the bowel. Although the other varieties are usually asymptomatic, mild symptoms of obstruction occasionally develop. Growths producing symptoms should be removed.

**Malignant Tumors** — Excepting the degenerative changes which may occur in polypoid coli, the malignant bowel tumors of childhood are almost universally sarcomatous. These rare neoplasms grow along the bowel wall rather than into the lumen and often attain large size. The majority are lymphosarcoma, round or spindle-cell types are occasionally reported. Cystic degeneration occurs commonly.

**Symptomatology** — The usual symptoms are those of the development of a mass with or without mild symptoms of obstruction. In the early stages, the tumor is often freely movable, it may be smooth

or irregular and at times quite tender. Metastatic dissemination occurs early accompanied by fever and cachexia.

**Treatment**—An early tumor in the absence of roentgenologic evidence of pulmonary metastases should be removed. In late cases radiotherapy is helpful and may prolong life. Repeated blood transfusions are supportive.

## DIVERTICULITIS OF THE COLON

Diverticuli are never found in the normal embryonal hind gut and when present are always acquired. They seldom develop during childhood and are rarely subject to inflammation. Although any portion of the colon may be involved the sigmoidal or cecal regions are generally affected.

**Etiology**—Diverticuli develop on the bowel circumference at the points of penetration of blood vessels. Atrophy of the subserosal fat may be a contributing factor in the development of the projection also traction upon the appendices epiploicae from adhesions to adjacent structures.

**Pathology**—The herniating mucous membrane is at first covered by submucosa, muscularis and serosa, ultimately however the muscle thins and the remaining submucosa becomes the chief supporting structure. Bulbous types with a narrow neck are subject to inflammation. Fecal material entering the diverticulum may become inspissated and produce decubitus necrosis. This may result in peridiverticulitis and the production of an inflammatory mass or in perforation and peritonitis.

**Symptomatology**—The symptoms of diverticulitis of the cecum mimic those of appendicitis and depending upon the pathology may be acute, subacute or chronic. Sigmoidal diverticuli produce focal signs and symptoms in the left lower quadrant. Ulceration of the lining mucosa may produce melena. In chronic cases the roentgenologic findings after a barium enema usually exhibit colonic spasm with or without a demonstrable diverticulum.

**Treatment**—(a) **Acute Diverticulitis**—Diverticulectomy should always be performed promptly as the condition is especially subject to perforation. Following amputation of the pouch the treatment of the opening into the intestine will depend upon its size. Small ostia may be closed by simple inversion suture of the bowel edges. Large openings are best treated by approximating their edges in the longitudinal axis of the gut so as to obviate luminal constriction. The suture line of No. 1 chromic catgut should be reinforced by a continuous Lembert of No. 0 plain catgut. The lumen of the gut should always be tested following the plastic repair. In rare instances a lateral anastomosis may be required for adequate patency. Abscess cases require drainage.

more than one-half of the cases. The dilatation begins abruptly at the recto-sigmoid and extends upward for a variable distance through the colon. As the patients grow older the disease may gradually extend until the entire colon is affected. The pathology is seldom limited to the transverse colon, hepatic flexure or ascending colon. In exceptional cases the rectum and lower ileum share in the dilatation which at times may be enormous. Very rarely gigantism of the entire alimentary tract is exhibited. In addition to being dilated the colon is often elongated and the mesentery greatly thickened. Hypertrophy of the gut wall especially of the muscularis is almost universally present and the mucous membrane may be so thickened that the exaggerated folds appear like valves. Cases of long standing may develop ulceration.

Histologically both the circular and longitudinal muscle fibers exhibit hypertrophy and frequently there is leukocytic infiltration and generalized fibrosis. The mucous membrane is greatly thickened and infiltrated with connective tissue elements. The serosa is usually normal. The mesentery reveals a marked increase in vascular elements with extreme engorgement and enlargement of the lymphatics, the changes being definitely limited to the mesenteric zone supplying the megacolon. Decubitus ulceration may result from the pressure of hard fecal masses but perforation is uncommon.

**Symptomatology**—The two chief symptoms are continuous extreme constipation and marked abdominal distention. In severe types the constipation exists from birth, in others it develops insidiously; in practically all cases it is definitely evidenced by the third month. The constipation becomes progressive and in many instances great distention occurs during the first year. The bowels may not move for two weeks or even longer. Following treatment an enormous evacuation may occur. The stools are seldom scybulous and at times are diarrheal; the difficulty of evacuation appears to be dependent upon insufficient propulsive power. Due to enormous accumulations of feces and gas the abdominal distention often becomes so tremendous that even copious evacuations do not materially diminish it.

The distention is usually uniform and the abdominal circumference may be increased to several times the normal girth. The abdominal muscles become thinned and separated, the skin shiny and the superficial veins distended (Fig. 18a). Huge peristaltic waves may be seen accompanied by audible borborygmi. Strange as it may seem vomiting and pain rarely occur. Dyspnea may result from pressure against the diaphragm. Volvulus of the sigmoidal loop develops occasionally.

The relief afforded by cathartics and mineral oil is generally ineffective and the constipation remains obstinate. Routine installations of warm olive oil followed by irrigations are at times helpful.

In severe cases the sphincter may have to be dilated under anesthesia and the fecal mass spooned out. The general health may remain good for several years and mild cases may reach adult life. Absorption of the products of chronic stasis ultimately produces degenerative changes in other viscera, nutrition fails and emaciation becomes progressive



serial roentgenograms. The history of continuous obstinate constipation beginning in early infancy is strongly suggestive of megacolon.

**Prognosis.**—The prognosis of idiopathic megacolon is definitely unfavorable. Few patients attain maturity and the majority die before the age of five years from inanition, intestinal toxemia or some intercurrent infection as bronchopneumonia, peritonitis from perforation occurs but rarely.

**Treatment.**—Surgery is the ideal therapy for megacolon but unfortunately it is attended with high mortality. Advanced cases are notoriously bad risks. Children who improve under medical treatment should not be subjected to operation so long as their progress is satisfactory. In the case of infants and young children, the mothers should be cautioned as to the importance of securing daily evacuations through the aid of diet and the use of mineral oil and warm olive oil enemas.

In progressive lesions prompt surgery becomes indicated. Colectomy, either total or segmental, is usually necessary to effect a cure. In debilitated infants the operative hazard of primary colectomy is prohibitive and graded or single-stage procedures should be elected. The gravity of the pathology should be carefully explained to the parents.

**Choice of Operation.**—Before discussing graded operative procedures, it should be noted that colectomy in a healthy infant or young child is apparently attended with less shock than in adults. The surgical procedures of colopexy and coloplication are mentioned, only to be condemned; entero-anastomosis about the segmental megacolon, with secondary partial colectomy, has also proven unsatisfactory.

**Primary Cecostomy**—Whereas preliminary drainage should always be instituted when one-stage colectomy is elected, the procedure is optional in two-stage resections. Cecostomy is preferable to colostomy for several reasons—it may be performed quickly and safely, in addition to diverting the fecal stream it offers an ideal ostium for irrigation of the distal distended loop, and being situated on the right side, it is removed from the zone of secondary resection. By repeated irrigations both rectally and through the cecostomy the megacolon may be completely drained of all gases and retained feces. General improvement in the patient's condition usually follows. Blood transfusions are also decidedly helpful. When the patient's competency appears adequate, secondary colectomy may be performed.

**Colectomy.**—In early life, secondary colectomy following cecostomy is best performed in one stage with immediate anastomosis as young children are ill-suited to the tedious postoperative care required in the Mikulicz-Bruns technique. In older patients, how-



ever, exteriorization of the bowel is definitely preferable. It is a much safer method and is ideally suited to segmental pathologies.

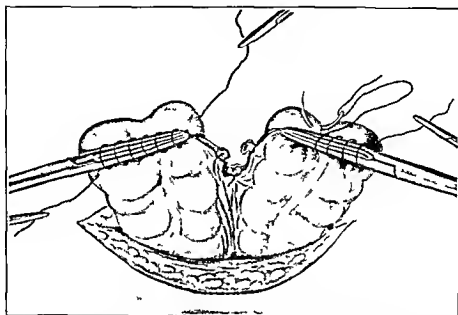


FIG 186 —The Kerr Parker basting stitch technique. The right angle stitch being applied with the loops passing over the clamp

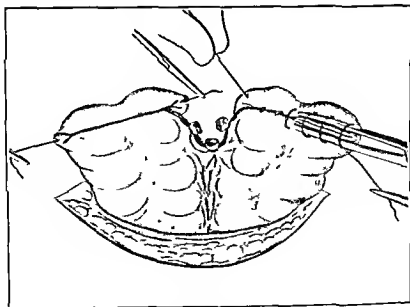


FIG 187 —Inversion and peritonealization of the divided ends of the bowel by withdrawal of clamps as the basting stitch is tightened

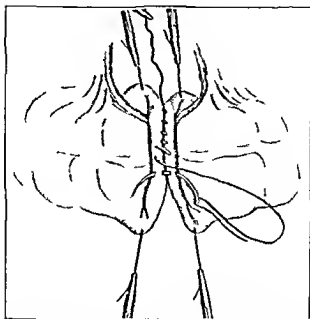


FIG 188 Anastomosis of sigmoid colon to the rectum, showing the blind bowel ends

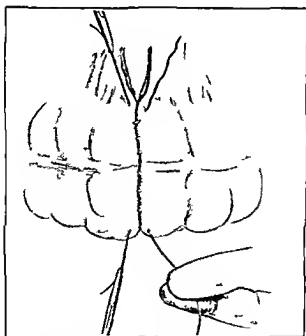


FIG 189 —Withdrawing the last suture after the anastomosis is completed. A second row of reinforcing sutures is then applied.

**Parker Kerr Technique** — The simplicity of the basting stitch in the Parker Kerr technic is an ideal method of performing an aseptic one-stage resection (Figs 186 to 189). After the bowel segment is withdrawn from the abdomen crushing forceps are applied in close apposition across the normal bowel above and below the megacolon. The intestine between each pair of clamps is divided by the antery knife and the mesenteric vessels are ligated so as to preserve adequate circulation in both loops. The basting stitch is a continuous right angled Cushing stitch the loop stitches passing over the clamp. The first and last sutures are applied under the clamp. The basting stitch is tightened as the clamp is removed thereby inverting the bowel edges with their peritoneal surfaces approximated. After both segments have been basted and inverted they are approximated by employing the ends of the basting stitches as guides. A primary suture of No. 1 plain catgut fused on its needle, is used to make the anastomosis. With completion of the anastomosis the basting stitches are withdrawn, the anastomotic area is then held between the thumb and index finger and by slight manipulation the previously basted agglutimized closures are opened thus restoring luminal continuity. A second row of continuous Lembert sutures is placed about the anastomosis after the mesenteric rent has been repaired. It cannot be overemphasized that a proximal vent for the escape of gas is a necessary safeguard in colonic resections. If a primary cecostomy has been employed it will ideally serve such purpose. Otherwise an appendicostomy or low ileostomy should be performed.

**Mikulicz Bruns Operation** — After the loop comprising the megacolon is withdrawn the opposing segments of the bowel are approximated by passing a few plain catgut sutures through the superior longitudinal bands and the abdomen is closed about the exteriorized bowel. On the third or fourth day or after ten days in the presence of a cecostomy the bowel is amputated with the enteric knife leaving two ostia side by side. At the end of two weeks a spur clamp is introduced by inserting one blade into each lumen. The clamp is gradually tightened each day. The intervening gut walls are thus cut through by slow necrosis luminal continuity being thereby restored. The roof may be closed under local anesthesia at a later date. In the hands of the average surgeon the two-stage Mikulicz-Bruns technic is a safer procedure than any other type of bowel resection.

**Total Colectomy** — The entire colon is very seldom involved in children. Such cases are generally of long standing and occur chiefly in adults. When present total colectomy may become necessary. In such instances the terminal ileum is anastomosed to the sigmoid (ileosigmoidostomy) and the colon may be removed at the same time or at a later period. The later procedure is safer

and is performed as follows. The terminal ileum is divided and both its ends are closed and inverted. The colon is then divided just above the recto-sigmoid junction. The clamp on the proximal colonic segment is allowed to remain and the distal sigmoidal ostium is closed and inverted. After the side-to-side ileo-sigmoidostomy has been completed the clamped end of the descending colon is brought out of the upper angle of the wound to remain as a fecal fistula for the discharge of mucus. Secondary colectomy may be elected at any future period.

**Postoperative Treatment**—Nothing should be given orally or rectally for the first five days. Some form of opium administered hypodermically is indispensable to prevent peristalsis. The saline-water balance should be well maintained by repeated hypodermoclyses or infusions of physiologic salt solution. The addition of 5 per cent glucose is advantageous; it is readily oxidizable and stimulates renal output. The artificial proximal vent is often life-saving as many leakages in colonic resection are the result of distention occurring on the third or fourth days.

**Lumbar Sympathectomy** Certain cures have recently been reported following lumbar sympathectomy. The left lumbar or both sympathetic trunks may be removed; in selected cases resection of the inferior mesenteric nerve or extirpation of the superior hypogastric plexus may be added. The approach is made either transabdominally or through bilateral lumbar extraperitoneal incisions.

## POLYPOSIS COLI

Polyposis coli is a disease in which single or multiple tumors grow from the wall of the bowel and project into the lumen. The condition presents two rather distinct types: (1) In which the tumors are single or few in number and are confined to one segment of the bowel; and (2) a diffuse polyposis of the entire rectum and colon; rarely the small intestine and stomach are also involved. The former variety occurs mostly about chronic ulcerative conditions in the adult colon; whereas the latter develops chiefly in children and young adults. Erdmann has emphasized this difference between the congenital or adolescent type and the adult variety.

The disease is uncommon and usually occurs in boys or young men from ten to twenty-five years of age. Cases have also been reported in infants. A familial tendency is sometimes noted.

**Pathology of the Adolescent Type**—The rectum is always involved and in some cases the pathology may arise there and spread upward to the colon. The masses projecting into the lumen of the bowel vary from minute elevations to the size of grapes or larger. Some are sessile, others pedunculated, and tremendous numbers may be present. They remain soft except when malignant.

degeneration occurs. A striking peculiarity is that carcinomas may develop in different tumefactions and at different times.

The tumors are generally adenomas or epithelial papillomas. Histologically the hypertrophied glands appear elongated, irregular and often cystic. The loose connective tissue stroma is supplied by a rich vascular network.

**Symptomatology** — The dominant symptom is that of intermittent diarrhea with profuse hemorrhage from the bowel. Secondary anemia may be pronounced. Upon proctoscopic examination the multiple tumefactions are readily palpable and visible in the rectum and sigmoid.

**Treatment** — Polyposis coli occurring in children is extremely difficult to cure and the various forms of treatment are essentially palliative. If the disease occurred segmentally, partial colectomy would be an ideal procedure. Unfortunately the pathology is almost universally diffuse and its delimitation cannot be determined.

Bleeding tumors within reach of the sigmoidoscope can at times be fulgurated or removed by electrolysis. In other instances radium or roentgen rays may cause shrinkage of the growths and control bleeding. Palliative appendicostomy or cecostomy, with subsequent through and through colonic irrigations is also of considerable benefit. Repeated blood transfusions are specifically supportive.

## CHAPTER XXXV

### THE RECTUM AND ANUS

#### CONGENITAL MALFORMATIONS OF THE RECTUM AND ANUS

ABNORMALITIES of the distal end of the gastro-intestinal tract were mentioned by the ancients. Paula Aeginetia in the seventh century described a successful operation for the relief of anal obstruction. A bistoury was plunged through the perineum into the rectum and the artificial anus was systematically dilated. This crude method was in vogue until Amussat (1835) recommended proctoplasty by careful dissection of the parts and suture of the rectal cul-de-sac to the anal site. Stromeyer (1844) advocated opening the pelvic peritoneum and exploring the pelvis for the rectum and about the same time the French school advised inguinal colostomy when perineal section failed. Bodenhamer (1860) wrote a comprehensive chapter on the classification and treatment of the various abnormalities and Cripps (1887) collected 100 operative cases and reported them. The writer published a review of 61 additional cases from which the statistical data in this chapter has been extracted.

**Embryology** —Recto anal defects result from faulty embryologic development during the first two months of fetal life. The rectum and anus develop separately, the former from the ectodermal and mesodermal layers of the blastodermic membranes and the latter from the epidermal. Whereas the lower end of the primitive intestine is at first connected with the neurenteric canal (postanal gut) this union soon disappears and the gut terminates in a cloaca common to it and the urachus. During the second fetal month the cloaca cavity is divided into an anterior and posterior portion by the urogenital membrane, the anterior becoming the urinary bladder and the posterior the primitive rectum or mesenteron (fig 190). Simultaneous with the descent of the mesenteron toward the perineum an infolding of epiblast termed the proctodeum occurs at the anal site. This depression extends inward to meet the blind rectal pouch into which it opens, the proctodeum thus forming the anus.

**Classification** —Failure of certain steps in embryonic development may result in the following malformations:

#### *A Atresia ani*

- 1 Partial occlusion or narrowing of the anus

- 2 Complete occlusion of the anus by a membranous diaphragm
- 3 Total absence of the anus, the rectum ending in a blind pouch
- 4 Total absence of the anus, the rectum opening into the bladder, urethra, uterus, vagina, perineum, or sacral region

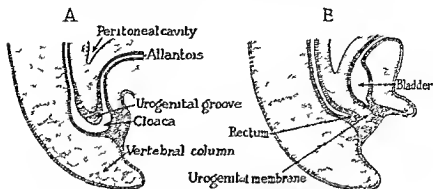


FIG. 190 Division of cloaca by urogenital membrane into bladder and rectum  
A 9-mm. embryo B 12-mm embryo

#### B *Atresia recti*

- 1 Partial occlusion or narrowing of the rectum
- 2 Complete occlusion by a membranous diaphragm
- 3 Complete absence or extensive obliteration of the rectum

#### C *Rectum and anus normal, but ureters, uterus or vagina emptying into the rectal cavity*

#### D *Absence of the large intestine, rectum and anus*

#### E *Rectal diverticula*

**Incidence**—Congenital malformation of the rectum and anus are of rare occurrence. Cripps, in a study of 78,000 births, found 17 cases of ano-rectal defects. Of 81,700 births at the New York Living-In Hospital, 17 presented malformations, and of 7239 births at the Manhattan Maternity Hospital there were 2 cases. These combined figures indicate that malformations occur once in every 4637 births. The sex ratio is 5 to 3 in favor of males. Anal defects are the most frequent and malformations with abnormal openings, *i.e.* vaginal or urinary, comprise the next most common group. The other varieties are extremely rare.

**Partial Occlusion**—In cases of partial occlusion of the anus or rectum the narrowing may be slight or of such degree as to scarcely admit the passage of meconium. The narrowing is usually annular, resembling a stricture formation. At times a considerable extent of the bowel is involved, as in a case reported by Cheever where the stenosed portion extended 18 inches the lumen being the size of a

goose quill (Narrowing of the recto-anal segment may also rarely result from fetal ulcerative proctitis and cicatricial contraction)

If the stricture is tight it will give rise to symptoms of obstructive ileus. The diagnosis is readily made by digital examination as the narrowing is near the anus. Mild cases may give no focal symptoms and reach adult life suffering only from obstinate constipation. Their congenital nature is evidenced by the absence of ulceration or other causative factors.

The treatment of partial occlusion comprises gradual dilatation with bougies supplemented when necessary by proctotomy. Palliative treatment should always be given a trial although division or excision of the stricture is often necessary for permanent benefit.

**Imperforate Anus**—In occlusion of the anus by a membranous diaphragm the obstructing membrane may be of variable thickness and firmness and be composed of either skin or mucous membrane. If the septum is thin bulging will occur when the child strains and such types occasionally rupture spontaneously. At times a small perforation is present which permits the escape of fluid meconium and thereby masks the condition until the feces become solid and obstruction develops. The diagnosis is self-evident upon digital or proctoscopic examination.

Treatment comprises crucial incision of the membrane. If the redundant flaps are dense they should be excised and the canal systematically dilated with bougies. This condition so amenable to treatment is unfortunately one of the rarest of anal malformations (Fig 191).

**Total Absence of Anus the Rectum Ending in a Blind Pouch**—This is the most common type of anal deformity. The rectal pouch may be normally situated in the sacral hollow lie loosely above the pelvic brim or be attached to some adjacent part. The perineum may be filled with cellular tissue or a distinct fibrous cord may extend downward to the anal site (Fig 192).

Many cases are overlooked at birth if an anal dimple is present and rectal temperature is not taken the absence of meconium attracts attention to the condition. Distention occurs late as the intestinal tract of the new born is sterile. Vomiting is also a late symptom. The patients generally succumb within ten days unless relieved by operation.

Opinion is divided as to whether operative interference should be attempted at once or after forty-eight hours. Delay has been recommended in order to give the rectum time to distend and become more prominent. This hypothesis is untenable as the meconium actually becomes less through absorption of its fluid content. Furthermore the intestinal tract is contaminated by the third day.

Although in approximately 90 per cent of cases the rectal pouch



is in the normal intrapelvic situs its actual position cannot be determined preoperatively. The presence of an anal depression is no indication of its proximity and bulging of the perineum on straining does not necessarily denote an intrapelvic rectum. Exploration of the bladder or vagina may be helpful, if either completely fills the concavity of the sacrum, the rectal cul-de-sac is probably high up. Abnormal narrowing of the distance between the tuberosities to less than 2 cm. is also presumptive evidence that the rectum is extrapelvic.

Wagenstien and Rice have proposed a roentgen-ray diagnostic test which is of value in determining the site of the cul-de-sac. If the child is held upside down by the lower extremities, the gas in

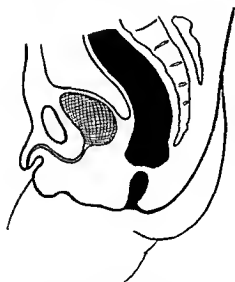


FIG 191 —Imperforate anus the anal canal being well formed (rare type)

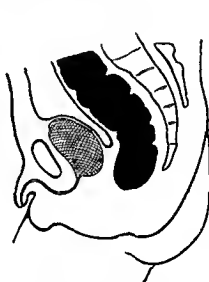


FIG 192 Imperforate rectum Anus absent (most common type)

the rectum will rise to the top and the bubble exhibited in a flat roentgen ray plate will indicate the distance between the rectal cul-de-sac and the skin of the anal dimple. The presence of a bubble also proves the absence of other intestinal deficiencies. The picture is best taken twenty-four hours after birth, by which time sufficient gas will have developed in the intestine.

**Proctoplasty**—The operation of proctoplasty is indicated unless there is strong evidence that the rectal cul-de-sac is extrapelvic. Light ether anesthesia should be employed. Good light and retraction are indispensable as the parts are very small. A sound or probe having been passed into the bladder as a guide, the patient is placed in the exaggerated lithotomy position with the thighs flexed

on the abdomen. The incision made along the perineal raphe from the tip of the coccyx to the scrotum or fourchet is cautiously deepened in the mid line by blunt dissection following the curve of the sacrum. Upon separation of the areolar tissue the distended greenish rectal pouch may be identified. The latter should be brought down and sutured by a double row of Lembert stitches to the cutaneous margin or to the external sphincter fibers when present. If there is difficulty in mobilizing the rectum the coccyx may be bisected or resected and the bowel sutured in its site. In the absence of sphincteric fibers control may be secured by Gersuny's procedure of axial rotation of the gut or muscle fibers from the gluteal region may be arranged in a figure-of-eight fashion about the bowel. The pouch may be opened at once as the meconium is sterile. In searching for the cul-de-sac dissection for a greater depth than 5 cm is unjustifiable. It is also unwise to open the pelvic peritoneum unless the pouch is visible just above. Blind puncture with a trocar is dangerous.

**Inguinal Colostomy**—Too much time should not be sacrificed in searching for the rectal pouch and the entire exploration should not exceed ten minutes. If unsuccessful a left inguinal colostomy should be performed (figs 193 and 194). Upon opening the abdomen the rectum may at times be sufficiently low to be grasped by dressing forceps and insinuated through the pelvic peritoneum to the anal site (celiotomy and combined proctoplasty). In rare instances the sigmoid is found on the right side.

The immediate results of proctoplasty are generally satisfactory. Sphincteric control however is seldom complete and the majority exhibit a variable degree of incontinence. Bougies must be systematically employed to prevent cicatricial contraction.

**Total Absence of Anus, the Rectum Opening Into the Bladder, Urethra, Uterus, Vagina, Perineal or Sacral Region**. This class comprises approximately 40 per cent of all cases. In females the rectum opens most commonly at the fourchet and rarely into the bladder or urethra (figs 195 and 196). In males however the opening frequently connects with the bladder or the urethra (figs 197 and 198). The communication with the bladder may be direct or by means of a narrow duct running through the bladder and opening into the bas fond between the ureteral orifices. In very rare cases where the bowel opens into the uterus the lower rectum as well as the anus is absent. Openings into the sacral or perineal regions by means of fistulous tracts occur rarely.

**Cases With Vaginal Communication**—Cases with a large opening into the fourchet may be asymptomatic and the condition is compatible with longevity and even conception. Morgagni reported a woman living a hundred years who bore several children and never knew of her abnormality. If the opening is small obstructive

symptoms develop when the feces change from a fluid to solid consistency. Many of these types have good sphincteric control and in early childhood are best treated by systematic dilatation.



FIG. 193 —Atresia recti with solitary right kidney



FIG. 194 — Appearance of colectomy four months later

Proctoplasty with an attempt at radical cure should be deferred until the parts are well developed, preferably at prepubescence, and should be resorted to only after systematic dilatation has

proved futile. A bent probe is passed through the vaginal opening and turned downward toward the perineum thus marking the end of the rectum. This is cut down upon by a median perineal incision.

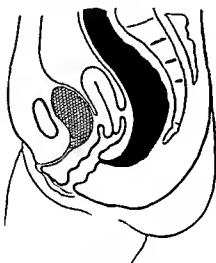


FIG. 195—Imperforate rectum with vaginal outlet

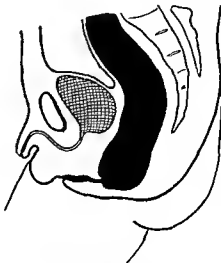


FIG. 196—Imperforate rectum opening in the perineum

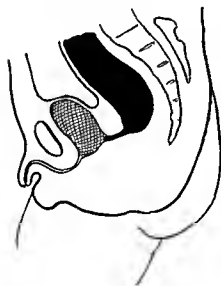


FIG. 197 Imperforate rectum with vesical outlet

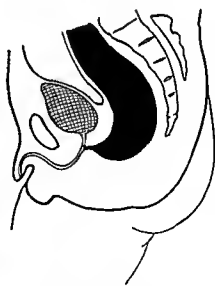


FIG. 198 Imperforate rectum with posterior urethral outlet

extending from the coccyx to the labial opening, the pubococcygeal fibers of the levator ani being carefully divided. The rectum is then thoroughly freed from the vagina until it can be placed without

The quantity of meconium passed is an index of the size of the fistulous opening. The condition is generally fatal early in life from the development of cystitis and ascending kidney infection. Nature is occasionally tolerant of such defects however and infection may not occur. When the communication is with the male urethra meconium is passed independent of micturition the first urinary flow may be meconium stained and the remainder clear. A large fistulous opening of this type is compatible with life and sexual potency.

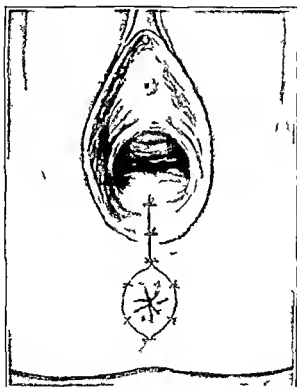


FIG. 200.—Replacement of anus well posterior only in front of coccyx.

Cases with vesical communication require early surgical intervention to prevent ascending kidney infection. The operation for radical cure is attended with such high mortality that inguinal colostomy is generally performed. This rarely results in closure of the fistula through divergence of the fecal stream. At a later date when the parts are well developed a proctoplasty may be attempted and the colostomy closed. The patient being placed in the lateral prone position with a catheter guide in the bladder a long median perineal incision is deepened through the levator ani

After the rectum has been isolated and freely dissected from the bladder, the vesical opening is freshened and carefully sutured. The rectum is then anchored at the normal anal site or to the sacral wound, and the parts allowed to heal by granulation. Continuous vesical drainage with irrigations should be maintained for several days.

**Partial Occlusion or Narrowing of the Rectum.**—This type is rare and occurs either about the level of the peritoneal reflexion or at the recto-anal junction (Fig. 201). In most cases the condition is the residuum of a perforated septum, being composed of a shelf of thick fibrous tissue lined above and below with mucous membrane. Occasionally multiple septa occur. The obstructive symptoms depend upon the size of the opening and fecal consistency.

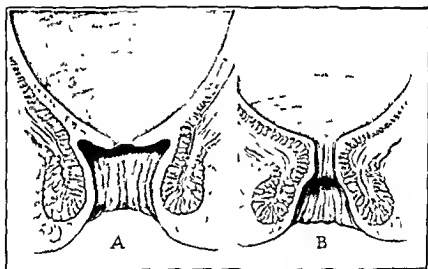


FIG. 201.—Congenital stricture of the rectum. A, Iris type, B, tubular stricture.

Although the presence of a normal anus may be misleading, the diagnosis is apparent upon proctoscopic examination. Treatment comprises crucial division of the septum, excision of the flaps, and systematic dilatation with bougies to prevent stricture formation.

**Complete Occlusion by a Membranous Diaphragm.**—These cases resemble the above type except that the septum is imperforate. The symptoms are those of complete obstruction, the diagnosis self-evident upon examination, and the treatment the same as the foregoing.

**Extensive Obliteration or Complete Absence of the Rectum.**—This deformity is almost always attended by an absence of the anus. Many cases of imperforate anus operated upon by the perineal route in which the rectal pouch is not discovered fall into this class.

The distal end of the rectum or the entire organ may be replaced by a fibrous cord. In such cases other signs of arrested development are usually present, the lower portion of the colon sharing most frequently in the defect. Colostomy offers the only life-saving procedure.

**Cases of Normal Rectum and Anus but With Ureters Uterus or Vagina Emptying Into the Rectal Cavity**—These are fetal curiosities and result from lack of normal development of the perineal partition. The condition is usually associated with other evidences of developmental arrest.

**Absence of the Large Intestine Rectum and Anus**—This condition occurs occasionally in monstrosities and the abnormal opening may be at the umbilicus in the thoracic wall or at some distant site even in the face.

**Rectal Diverticulæ**—These are extremely rare and all the coats of the bowel are involved in the herniation. They are prone to fill slowly with fecal material and becoming inflamed produce symptoms of focal pain and tenderness or upon rupturing of local or diffuse peritonitis. Although subject to diagnosis by proctoscopic examination or radiographic findings after bismuth injection, the few reported cases were discovered during exploratory laparotomy.

**Prognosis of Ano rectal Defects**—The prognosis depends chiefly upon the nature of the malformation and the condition of the patient. It is well recognized that congenital defectives have a lowered resistance. A factor insufficiently emphasized however is the not infrequent concurrence of multiple congenital defects, *e. g.* stenoses elsewhere in the gastro-intestinal tract, polycystic kidneys, etc.

In a case of atresia recti reported by the writer the necropsy findings were as follows. Chest normal. Upon opening the abdomen the stomach is markedly distended, filling upper two-thirds of abdomen. Lying about it are the collapsed loops of small intestine. Ascending, transverse and descending colon collapsed. Upon tracing the small intestine to the gastroduodenal junction a cord like stricture formation presents at the pylorus. Gas in the stomach cannot be forced into the duodenum. The stomach easily holds 11 ounces of water. Stenosis at the pylorus is complete involving both mucosa and muscularis. Liver, Gall bladder and cystic duct are absent. There is a rudimentary fissure for the gall bladder in front of the right end of the portal fissure, the quadrate lobe being imperfectly marked off from the remainder of the right lobe. To the left it is connected with the left lobe by a well marked pons hepatis. The common bile duct is formed by the union of the two hepatic ducts and occupies its normal position in the free edge of the gastrohepatic omentum. There is no dilatation of the common duct and no intrahepatic gall bladder is present. There is no evidence of pelvic peritonitis. The pelvic peritoneum adheres to the

After the rectum has been isolated and freely dissected from the bladder the vesical opening is freshened and carefully sutured. The rectum is then anchored at the normal anal site or to the sacral wound and the parts allowed to heal by granulation. Continuous vesical drainage with irrigations should be maintained for several days.

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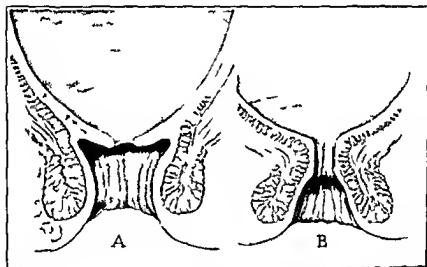


FIG. 201. Congenital stricture of the rectum. A Iris type. B tubular stricture.

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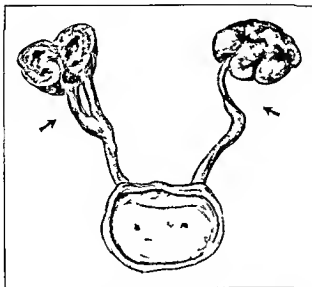


FIG 202 Atresia recti Both kidneys polycystic with thickened tortuous partially stenosed ureters (Brenner courtesy of Surg Gynec and Obst )

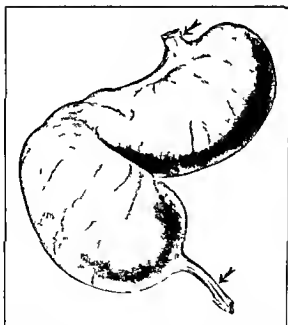


FIG 203 —Same case Greatly distended stomach containing 11 ounces of fluid Lower arrow indicates stenosed duodenum Gall bladder absent (Brenner courtesy of Surg Gynec and Obst )

rectum whose lower edge is held to the pelvic floor by sutures. The pelvic rectal space is clean. Left kidney multilocular, the size of a plum. Upon section it is polycystic and there is little cortical tissue. Two pelves unite in a common tortuous ureter which presents five incomplete strictures. Right kidney multilocular, slightly smaller and polycystic. Cysts contain purulent material. Ureter is distended and impervious in its lower third. Diagnosis: atresia recti, pyloric stenosis, absence of gall bladder, polycystic kidneys, strictures of both ureters." (Figs. 202 and 203.)

Clinically, the cases of ano-rectal malformations fall into two classes: (1) Imperforations with complete obstruction requiring immediate operative interference and (2) those with fistulous openings which are subject to palliative treatment. In the former the optimum indication is to establish an outlet that will imitate the natural anus in both function and position.

A summary of 61 cases studied by the author is appended in Tables 1 and 2.

TABLE 1—SUMMARY OF CASES

Types	Cases	Male	Female	Not stated	Operated	Successful	Died (causes)		Unoperated	Surgical mortality per cent
							Surgical	Non surgical		
Atresia ani	27	17	6	4	12	17	5	5	0	18.5
Atresia ani: vulvar outlet	10	0	10	0	4	4	0	0	0	0
Atresia ani: complete occlusion	3	2	1	0	2	2	0	0	1 <sup>1</sup>	0
Atresia ani: perineal outlet	2	1	1	0	1	1	0	0	1 <sup>1</sup>	0
Atresia ani: scrotal outlet	2	2	0	0	2	2	0	0	0	0
Atresia recti	12	9	1	2	11	3	4	4	1 <sup>1</sup>	33.3
Atresia recti: vaginal outlet	3	0	3	0	3	2	0	1	0	0
Atresia recti: urethral outlet	7	1	1	0	2	0	2	0	0	100.0
Totals	61	32	23	6	52	31	11	10	9	21.1

<sup>1</sup> Died      <sup>1</sup> Refused

TABLE 2—TYPES OF OPERATION

Operation	Cases	Successful	Died (causes)		Surgical mortality per cent
			Surgical	Non surgical	
Perineoplasty (mostly proctoplasties)	23	19	6	4	20.0
Inguinal colostomy	11	2	4	5	36.4
Perineal dissection for fistulous openings	10	9	0	1	0
Celostomy and proctoplasty	2	1	1	0	50.0
Totals	52	31	11	10	21.1

The causes of death are tabulated as surgical and non-surgical. The latter includes cases in which there were concomitant congenital

conditions incompatible with life or in which non-surgical complications supervened. Of the 52 operative cases 31, or 60 per cent, terminated successfully. Unfortunately, the records of several cases are incomplete and give no data as to functional competency of the newly formed anus. The majority of those noted, however had complete or partial control. Of the 21 remaining cases, 10 died of non-surgical complications, while 11 succumbed to the immediate or remote result of operative interference, giving a surgical mortality of 21.1 per cent. In both tables the death rate rises in direct ratio to the severity of the malformation and the following conclusions appear justified. Operations for atresia ani with simple occlusion or with fistulous openings into the vulva or perineum are attended with little danger; the correction of atresia recti with vaginal communication is a relatively safe procedure; the mortality of cases with complete atresia ani is approximately 18.5 per cent; and when accompanied by rectal defects rises to 36.3 per cent; inguinal colostomy is lethal in 36.4 per cent and should therefore be recommended only as a method of necessity, the relief of atresia recti with urethral outlet is attended with formidable mortality.

### TUMORS OF THE RECTUM.

**Adenoma.**—Adenomas are the most common variety of benign rectal growths. They occur rather frequently in early life, especially between the ages of two and five years.

The tumors may be single or multiple, sessile or pedunculated, and vary in size from a pea to a large cherry. The most common type is a solitary pedunculated growth covered with mucous membrane similar to that lining the bowel. Less often the tumor is finely trabeculated and its raspberry-like surface may be dark red and covered with flecks of blood. The growths occur at any level of the rectum and when multiple may involve the colon.

**Pathology.**—The origin of the tumors, in common with those occurring in polyposis coli, remains obscure. Many observers place them in a class between inflammatory growths and true neoplasms. Although their occasional presence in chronic ulcerative conditions of the bowel suggests the former, in most instances the tumor is the sole pathology. Whereas rectal adenomas in adults are subject to malignancy, those in children are rarely so.

The growths appear to originate in the mucosa and consist of normally arranged glandular epithelium in a supporting structure of connective tissue. In pedunculated types the stalk grows from the submucosa and consists of connective tissue and blood-vessels. The consistency and vascularity of the tumors vary according to their relative connective tissue content. Ulceration and bleeding occur commonly from irritation and futile attempts at extrusion.

**Symptomatology** —The most striking symptom in young children is the passing of a small amount of bright red blood and mucus at stool accompanied by straining and constipation. When the growth is situated high in the rectum, the blood is occasionally dark and clotted. Bleeding between evacuations occurs but rarely. Low-situated polypoid growths may be extruded through the anus with each movement and prolapsus recti may result from the pedicle drag.

**Diagnosis** —Most adenomas occur in the lower rectum and are palpable upon digital examination as soft elastic or firm tumefactions which can often be expressed through the anus. Tumors situated at higher levels are readily demonstrable by proctoscopic examination. *In all cases of bleeding from the rectum adenoma should be strongly suspected.*

**Treatment** —Adenomas should always be removed when possible. Pedunculated tumors may be treated as follows. Following dilatation of the sphincter the growth is grasped with an Allis clamp and retracted toward the anus. The pedicle is then ligated with silk as close as possible to the rectal wall and the tumor amputated. A thickened pedicle is best secured by a transfixion ligature in order to prevent slipping and secondary hemorrhage. Growths situated high in the rectum may be removed with an electric snare. Sessile varieties should be widely excised followed by cauterization or electric coagulation of the base. When both the rectum and colon are involved by multiple polypi and fulguration of the bleeding tumors is unsuccessful diversion of the fecal stream through either a cecostomy or appendicostomy may control the hemorrhage. (Refer to Polyposis Coli.)

**Fibrous Polypi** —Fibrous polypi of the rectum are comparatively rare in children. They occur at the mucocutaneous line in association with such conditions as fistula, chronically inflamed hemorrhoids and prolapse of the rectum.

**Pathology** —The growths vary in size to 1 cm. or more in diameter and at times are multiple. Pedunculated types predominate. The tumors are covered with squamous-cell epithelium and the stroma consists of fibrous tissue which often exhibits dense round-cell infiltration.

**Symptomatology** —Protrusion at stool of a small firm mass which reduces spontaneously is generally the only symptom. Pain may occur if the surface of the tumefaction becomes ulcerated.

**Treatment** —This comprises removal of the growth well down to its point of attachment, and correction of the etiologic condition.

**Papilloma of the Rectum (Villous Tumors)** —Papillomas of the rectum are rare. The growths may be lobulated or villous and usually spring from a broad base. They are red in color, of soft consistency and may attain large size.

**Pathology**—The tumors consist of myriads of connective tissue branches covered with high columnar epithelium. Adenomatous elements may occur at the base and the acini are often cystic.

**Symptomatology** The chief symptom is bleeding from the bowel often accompanied by a profuse mucous discharge. Low situated tumors may prolapse through the anus at stool.

**Treatment** The growths should be completely removed or destroyed as many papillomas are potentially malignant. In excising low growths a small surrounding area of healthy mucosa should be removed with the tumor. High growths are best treated by fulguration.

### PROLAPSE OF THE RECTUM

This condition of abnormal descent into the rectum and protrusion through the anus of one or more coats of the bowel occurs frequently in children. It is especially common early in life and over 60 per cent of the cases develop between the ages of one and three years.

**Etiology** The etiology is not entirely clear. The unusual length of the intestine and its weak fixation in early life combined with the diminished anterior concavity of the sacrum and relative high position of the bladder and uterus appear to be definite predisposing factors. The usual exciting cause is straining at stool from such conditions as constipation, diarrhea, polypi, worms, phimosi and gravel. Compelling a constipated child to sit for long periods on a high toilet seat is a common error. The condition may also develop in wasting diseases from absorption of the fatty cushions which normally support the rectum and from excessive vomiting or coughing.

There are three types of prolapsus recti: (1) Mucosal or partial prolapse, (2) complete prolapse, and (3) prolapse of a sigmoid rectal intussusception. The first type is of frequent occurrence, the second very uncommon, and the third exceedingly rare.

**Mucosal or Partial Prolapse of the Rectum**—Partial prolapse is a protrusion of the rectal mucosa through the anus and is an exaggeration of the physiologic extrusion occurring at defecation. The mucous membrane, loosely attached to the underlying structures by fibrous and elastic tissue, is normally retracted after defecation. In pathologic conditions the tissues lose their elasticity and become elongated, and the mucosa is protruded to an abnormal degree, the extreme limit being about 2 inches. The prolapse usually involves the entire circumference of the bowel and consists solely of mucous membrane (Fig. 204). The sphincter muscles remain normal.

**Symptomatology** Prolapse of a ring of mucosa during defecation is in most instances the only symptom observed. The protrusion develops gradually and generally recedes spontaneously. In protracted cases the membrane may ultimately remain extruded.

Rarely an acute prolapse develops which may become strangulated unless promptly reduced

The color of the prolapsed ring of mucosa is at first normal but after repeated or protracted extrusion it becomes bright red. Secondary inflammatory changes may result in ulceration and the discharge of mucus, pus and blood. An associated pruritus is not uncommon.

**Diagnosis** —In partial prolapse the entire protrusion is covered by mucous membrane, whereas in complete prolapse the skin is continued onto the prolapse at its anal exit to join the mucosa at the mucocutaneous line. Furthermore, in partial prolapse the

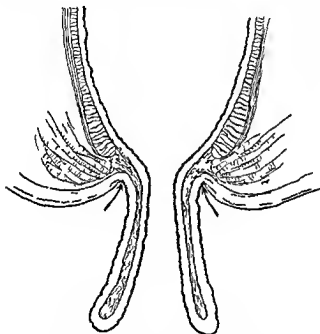


FIG. 204 —Partial prolapse of the rectum. Arrows indicate the mucocutaneous junction; the protrusion consisting only of mucosa.

mucosal folds radiate from the lumen toward the circumference while in the complete type they are arranged concentrically around the bowel.

**Treatment** —Removal of the exciting cause will cure the majority of cases and operative procedures are rarely necessary. Regulation of the bowels through proper diet is the first essential and the child should not be allowed to sit at stool for more than the required period of defecation. At times the prolapse may be prevented by pressing the buttocks together at the time of bowel movement. Failing in this, the prolapse should be reduced immediately and the child kept in the recumbent position for ten minutes. If protrusion occurs

the mucous membrane and completely severs the mucosa from the skin. The friable mucous membrane is elevated by blunt dissection to the apex of the prolapse care being taken not to injure the sphincter muscle which lies directly under the submucosa at the mucocutaneous line. Troublesome bleeding is controlled by meticulous hemostasis. The denuded muscularis of the prolapsed segment is then divided into four sections by inserting four No. 1 chromic sutures beginning at the mucocutaneous line and picking up three or four bites of muscularis and ending at the apex of the

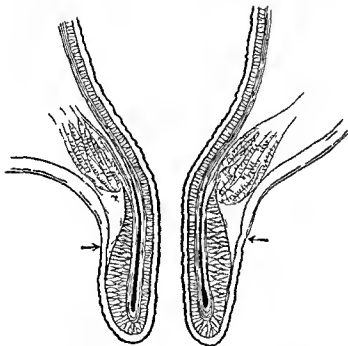


FIG. 205 — Complete prolapse of the rectum. Arrows indicate the mucocutaneous junction. The prolapse consists of the entire rectal wall.

prolapse. Upon tying the sutures the prolapsed segment becomes collapsed and slips back into the pelvis above the sphincter muscle. Should the latter be atrophied from overstretching mattress sutures may be inserted into one or more quadrants of the muscle. The mucosa which was denuded from the prolapsed segment is then cut away and the remaining mucosal cuff is sutured to the skin thereby reestablishing the mucocutaneous junction.

**Sigmoid rectal Prolapse** — In the very rare condition of sigmoid rectal intussusception several inches of the bowel may protrude from the anus.



**Treatment** Laparotomy is performed through a lower left para-rectus incision. After reduction of the intussusception and obliteration of the cul-de-sac of Douglas the pelvic colon is mobilized and sutured to the psoas muscle.

## HEMORRHOIDS

Hemorrhoids seldom develop in early life. Although in cases of bleeding from the rectum their possibility should always be considered it will usually be demonstrated that the hemorrhage is due to other causes, most commonly a polypus or rectal prolapse.

**Varieties**—Hemorrhoids may be external or internal or combined extero-internal. The former originating as varicosities in the inferior hemorrhoidal veins below the mucocutaneous line and having a cutaneous covering occur but rarely; the internal developed from radicles of the superior hemorrhoidal vein above the mucocutaneous line and covered by mucosa comprise the usual type.

**Etiology** Chronic constipation is the dominant factor in the development of hemorrhoids. The rectal mucosa is loosely connected with the muscularis and the loose submucal areolar tissue in which the venous radicles traverse offers little resistance to the development of varicosities in the presence of passive congestion.

**Symptomatology of External Hemorrhoids**—The tumefactions are subject to thrombosis from strain or traumatism. Through rupture of the venous radicle the pile becomes distended with blood and presents as a shiny, livid, tender swelling at one side of the anal verge. The condition is usually accompanied by acute pain and tenesmus. Palliative treatment comprises the application of lead and opium or 5 per cent magnesium sulphate dressings. Unless the condition rapidly subsides an ellipse of skin over the dome of the tumefaction should be excised and the clot evacuated. Healing progresses more rapidly and satisfactorily without drainage or sutures.

**Symptomatology of Internal Hemorrhoids**—The painless vascular tumors are situated just above the ano-rectal line, most commonly in the two posterior and right anterior quadrants of the rectum. At times a fourth pile occurs in the left anterior quadrant or occasionally in the central posterior region. The tumefactions are readily seen upon proctoscopic examination.

The pile consists of dilated radicles of the superior hemorrhoidal vein and a few unchanged arterioles supported by connective tissue stroma and covered with rectal mucosa, more or less diseased. Slight traumatism may rupture the vessels and lead to ulceration and hemorrhage. The latter generally attracts attention to the condition although in some instances the hemorrhoids may prolapse during defecation. The complications of fissure, fistula or ischio-rectal abscess seldom develop in early life.

**Treatment** —Palliative treatment is always indicated unless the condition is well developed. The relief of constipation through appropriate diet and mild laxatives will generally stop the bleeding and effect a cure. The bowels should be regulated by limiting the protein intake and adding green vegetables and fruits. Agar-agar and the mineral oils are the best laxatives. The injection of 1 ounce of olive oil into the rectum at bedtime is often efficacious in lubricating the canal and softening the stool. Pure ichthylol applied to the hemorrhoid will usually check the oozing of blood. A blunt-tipped glass rod makes a convenient applicator.

**Hemorrhoidectomy** —Advanced cases of internal hemorrhoids and those complicated by prolapse or strangulation should be subjected to hemorrhoidectomy. Although the injection treatment of uncomplicated internal hemorrhoids in adults is curative in many instances, the procedure is rarely adaptable to children. Surgical removal may be performed by either the open or closed method, the former being typified by the clamp and cautery, and the latter by ligation and excision.

Under general anesthesia the sphincter ani is gently dilated to a slight degree. Rapid and extreme dilatation may produce division and hemorrhage into the muscle with resulting fibrosis and dysfunction. In most instances three vascular tumors are demonstrable. Each is treated separately according to one of the above-mentioned methods. Although the popularity of the clamp and cautery technic is warranted by its simplicity of performance and excellent after results, the open ligature method is preferable in children.

**Ligature Method** —An Allis clamp is applied to the outer end of each hemorrhoid and fanwise traction on them gives an excellent exposure. When two hemorrhoids coalesce they are treated as a single tumor. The most dependent pile is removed first in order to have the operative field unobstructed by oozing. While the pile is elevated the mucocutaneous margin is incised with scissors. The hemorrhoid is then dissected up by dividing the mucous membrane part way on each side until only a pedicle remains which contains the central artery and vein. A transfixing ligature of linen or silk is then applied to the pedicle as high as possible and the pile is amputated, leaving a sufficient stump to hold the ligature. (Fig 206.) When more than one hemorrhoid is removed, it is important to leave a strip of mucosa between the piles connecting the anal skin with the rectal mucous membrane. If this is omitted ulceration and delayed healing may lead to stricture formation. A No. 20 F soft rubber catheter, smeared with vaseline, is inserted into the rectum for about 3 inches and gauze dressings are packed snugly about both sides, the catheter being held in place by a transfixing safety pin.

The use of the rectal catheter offers certain definite advantages

Should postoperative bleeding occur it will be recognized at once flatus is readily expelled and an enema may be administered painlessly through the tube on the third day. A warm mixture of milk and molasses is especially soothing and effective and the catheter is usually expelled with the evacuation. After treatment comprises regulation of the bowels.

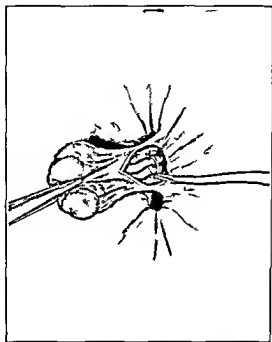


FIG. 206. Ligation of hemorrhoidal vessels.

### FISSURE IN ANO

Fissure in ano is a longitudinal rent of the mucocutaneous lining of the anal canal characterized by intense pain during and after defecation. Although essentially a disease of adult life its occasional occurrence in childhood warrants recognition. Kjellberg found fissures in 128 of 9098 children examined at the Stockholm Polyklinik (1.4 per cent).

**Etiology.** The anal canal with its delicate mucocutaneous lining is poorly supplied with blood and any wound or excoriation may eventuate in fissure formation. The exciting cause is trauma most commonly from the passage of a hard scybulum and occasionally from a sharp foreign body as a fish bone or the insertion of an enema tip.

**Pathology** —In over 90 per cent of the cases the fissure is situated between the radiating folds at or near the posterior anal commissure and generally below the level of the anal valves. The elongated crack like appearance of the lesion results from compression of its sides by the sphincter muscles. When the latter are relaxed the fissure is demonstrable as an oval or round ulceration. Although at first superficial the process may gradually extend through the mucosa to the sphincter muscle and through the deposition of fibrous tissue eventuate in the so-called chronic irritable ulcer. The latter is frequently accompanied at its lower angle by a skin tag or sentinel pile pathognomonic of fissure. This stage of chronicity however is rarely seen in childhood. Infection from a fissure to adjacent tissues may result in abscess formation and subsequent fistula.

**Symptomatology** —Pain the dominant symptom is often excruciating in character and out of all proportion to the insignificant size of the lesion. The child becomes afraid to move its bowels and screams during the act of defecation. Occasionally a drop of bright blood is passed with the stool or the latter may be blood streaked.

The diagnosis is self-evident upon careful examination of the anal canal. In most instances this may be performed painlessly if the child is placed in the Sims position and the anal structures are exposed by gently spreading the buttocks. Anesthetic powder blown upon the parts is at times helpful. An intractable patient may require brief general anesthesia. Digital examination is next performed to detect any complicating pathology. Complete exposure of the fissure is then obtained by introducing a small speculum such as the Gorsch and placing the slide toward the fissure.

**Palliative Treatment** —Success in the therapy of fissure depends upon rest and drainage. The first essential is to obtain soft daily evacuation of the bowels through the administration of a bland diet supplemented with agar agar and mineral oil. Two to 4 ounces of warm olive oil may be injected into the rectum through a catheter before each movement. Leaf and opium wet dressings or a hot Sitz bath often relieve the pain following defecation. In the early stages the application of pure ichthyol is frequently curative. Three per cent silver nitrate solution is also recommended. If a drop of 10 per cent cocaine is applied first the topical application may be made with only slight discomfort. Powdered orthoform or anesthetic may also be effective in very painful lesions. Undermining the lesion with either quinine and urea hydrochloride or eucaine is seldom practical in young children. Chronic ulcerations and cases accompanied by a sentinel pile are not amenable to palliative treatment.

with a sharp scalpel from the upper end of the fissure through its base and out onto the skin for at least 1 cm the external sphincter being thereby partly divided (Fig 207) The incision should be carried sufficiently deep to form a smooth groove that will readily admit the finger into the rectum The mucosal and anal margins of the fissure are then excised and a vaseline gauze drain is inserted The bowels are moved by an oil enema on the third day and healing is usually complete by the tenth

## ANO-RECTAL FISTULA

An ano rectal fistula may be complete or incomplete In the former there is a pathologic communication between the skin and the anal or rectal canal and in the latter the tract has an orifice at one end only The term blind internal fistula designates an incomplete fistula the ostium being in the ano rectal mucosa and

blind external fistula implies the sinus opens only on the skin Since the successful treatment of fistula depends largely upon locating the internal opening the following classification is of clinical value Anal opening in the anus ano rectal opening at the pectinate line between the internal and external sphincter and rectal opening in the rectum

**Etiology** —Although fistulae occur chiefly between the third and fifth decades children are not infrequently affected The condition is almost invariably a sequela of abscess formation and the sequence of events is generally as follows The passage of a hard stool or at times a sharp foreign body produces a fissure of the ano rectal mucosa and infection therefrom to the submucosal and adjacent tissues results in abscess formation Rupture of the latter occurs spontaneously through the skin into the bowel or in both directions and the contracted wall of the abscess cavity forms the fistulous tract

**Pathology** —The fistulous tract is lined with granulations and surrounded by fibrous tissue The suppurative process which eventuates in its formation usually develops in the triangular space behind the anus If rupture occurs through the fissure a blind internal fistula results More often however the abscess points externally through the skin at or near the mid line producing either a complete fistula or a blind external fistula (Fig 208) When the skin is perforated on both sides of the raphe the condition is termed a *horseshoe fistula* Cutaneous ostia are prone to close and seal the opening When this occurs a secondary abscess may form and perforate the skin at another site Multiple external openings may thereby be produced

The infection extends at times into the ischio-rectal fossa and the resulting abscess limited above by the anal fascia and externally

by the obturator fascia points toward the skin (Refer to Ischio-rectal Abscess) If allowed to rupture spontaneously, the contracting walls of the abscess cavity are apt to produce a tortuous fistulous tract

Although 15 to 20 per cent of fistulae in adults are tuberculous, the infecting organisms in childhood are almost universally the cocci and bacilli ordinarily composing the intestinal flora. The failure of healing in complete and blind internal fistulae results from continuous reinfection through the ano-rectal ostium. Persistence of blind external types may be due either to an undetected portal of entry in the rectal mucosa or to a densely fibrotic tortuous tract

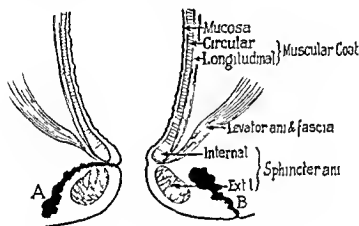


Fig. 208. A Blind internal fistula B Blind external fistula

inserted into the rectum and the solution is then injected through the external fistulous ostium. A blue stain on the applicator indicates the presence of a complete fistula. (Fig. 209.)

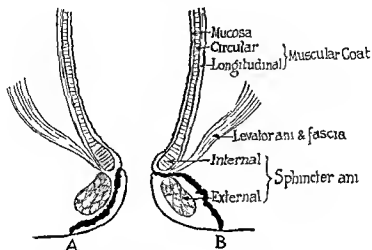


FIG. 209 — *A* Complete subcutaneous-submucous fistula. *B* Complete fistula passing laterad to the external sphincter.

**Treatment** — Although in rare instances an early fistulous tract may be cured through the palliative injection of escharotics such

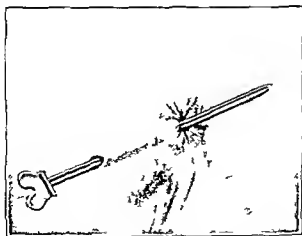


FIG. 210 — Probe passed through a fistula before laying open the tract.

as Cutler's solution, 95 per cent carbolic or 5 per cent silver nitrate, the safest and most satisfactory treatment is incision and drainage. The elastic seton ligature is mentioned only to be condemned.

**Operation**—This comprises incision and drainage of the fistula so as to include the opening into the bowel and all collateral branches of the tract. Under ether anesthesia, the patient is placed in the lithotomy position and the sphincter ani is gradually and moderately dilated. When an external ostium occurs, a speculum is introduced into the rectum and the fistulous tract is injected with methylene blue peroxide solution to determine the site of the internal opening. A probe or grooved director is then passed through the fistula and brought out of the anus and the intervening structures are divided. (Fig. 210.) While the margins of the wound are retracted by Allis clamps, all tissues stained by methylene blue are trimmed away, including the skin edges about the external opening. This is followed by the application of 95 per cent phenol, neutralized with alcohol. A catheter is then inserted well into the rectum and the wound is packed with sterile vaseline gauze strips. The dressings are changed daily, the bowels being moved on the third or fourth day by a warm oil enema injected through the catheter. If the fistula has been thoroughly laid open and properly drained, permanent cure will generally occur.

### CRYPTITIS.

The semilunar valves between the columns of Morgagni form minute pockets opening upward, termed the crypts of Morgagni. Normally they are quite small and scarcely demonstrable. When overdeveloped, small particles of feces or foreign bodies may become impacted in them and produce inflammation. The cryptitis may subside, or eventuate in ulceration and abscess formation and terminate in a submucous fistula.

Many cases of cryptitis in early life are probably overlooked as the condition occurs more commonly than the literature would indicate. In the absence of acute pain and tenesmus, the symptoms of itching or anal irritability are apt to be attributed to worms or irritating stools. Digital examination is often negative and unless a careful proctoscopic examination is made, the pathology may be missed. The writer recently examined a child, aged four years, who had suffered from tenesmus and pruritus ani for two months due to an impacted fruit seed in an anal crypt. (Fig. 211.) Complete relief followed its removal.

**Symptomatology.**—Depending upon the acuity of inflammation, the symptoms may vary from lancinating pain and tenesmus to those of itching and constipation, and occasionally reflex dysuria. Digital examination may detect an exquisitely tender focal spot or be entirely negative, especially if a submucous sinus has formed. Under good illumination and through the aid of a fenestrated proctoscope, each crypt should be examined by a shepherd's crooked



probe. Normal crypts are relatively insensitive and acute tenderness indicates inflammation. If a sinus has formed the probe will readily enter it.

**Treatment**—Simple inflammatory cryptitis may be cured through the daily application of pure ichthyol to the crypt. A foreign body requires removal. If a fistula has developed it should be ablated by dividing the overlying tissue with a bistoury after a probe or grooved director has been inserted into the tract.

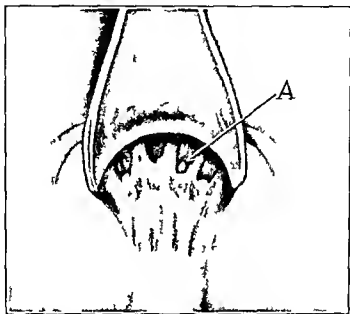


FIG. 211.—Seed in a crypt of Morgagni producing intense tenderness.

## PERIANAL PERIRECTAL AND PELVIRECTAL ABSCESS

Abscesses about the anal canal and rectum occur quite frequently in children. Their early diagnosis and treatment are especially important because of the tendency to fistula formation.

**Etiology** Infection occurs most commonly through the anorectal mucosa, the primary pathology being a fissure ulcer or cryptitis. It may also begin in the perianal hair follicles or sebaceous glands and infrequent causes include perforation of the mucosa by a sharp foreign body, as a fish bone tears from the rough insertion of an enema nozzle, ulceration of internal hemorrhoids and direct trauma. The pelvirectal spaces may be involved secondarily from a pelvic abscess or rarely from caries of the lower vertebrae or pelvic bones.

**Pathology**—Lymphatic extension of the primary infection into the cellular tissues results in abscess formation. Cultures there-

from generally exhibit a mixed infection of staphylococci streptococci and *B. coli* the latter occurring probably as a complicating rather than a causative organism. The presence of gas in the abscess is more commonly due to gas forming organisms than to a communication with the bowel. Tuberculous abscess secondary to pulmonary or intestinal tuberculosis is very uncommon and primary tuberculosis is exceedingly rare.

Depending upon anatomic situation the abscesses may be classified as follows:

1. Infralevator abscess
  - (a) Cutaneous
  - (b) Marginal
  - (c) Ischio-rectal
2. Supralevator abscess
  - (a) Retro-rectal
  - (b) Superior perirectal
  - (c) Interstitial (or mural)

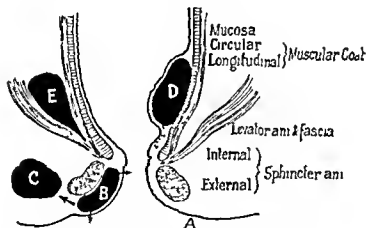


FIG. 219.—Various types of abscesses. A Perianal B marginal C ischio-rectal D submucosal E supralevator

**Infralevator Abscesses**—**Cutaneous Abscess**—The condition results from infection of the perianal hair follicles or sebaceous glands. Although essentially a folliculitis the subcutaneous tissues may become secondarily involved. Coagulation and sterilization of each furuncle with the endotherm needle is perhaps the best form of treatment. A common practice consists of cleansing the parts with alcohol and puncturing each abscess with a sharp toothpick previously dipped in 95 per cent carbolic acid and applying hot boric acid compresses. Incision and drainage is less satisfactory and may spread the infection.

**Marginal Abscess** — The process is a circumscribed collection of pus just beneath the skin at the anal margin. The infection usually results from a fissure and the abscess commonly develops postero-lateral to the anus. The pus tends to burrow upward beneath the mucocutaneous lining of the anal canal and unless evacuated may rupture in one of three ways: by drainage through the fissure resulting in a blind internal fistula; through the skin producing an external fistula; or spread beneath the fissure to the ischio-rectal fossa.

**Symptomatology** — The inflammatory process generally develops suddenly and the onset may be accompanied by a chill followed by pyrexia and malaise. A localized swelling is soon manifested

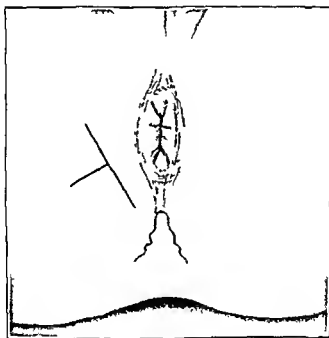


FIG. 213. Correct incision for opening a perianal or ischio-rectal abscess.

postero-lateral to the anus. In unusual cases the abscess develops entirely within the anal canal and is only demonstrable by digital examination. A slow developing abscess suggests tuberculosis.

**Treatment** — This comprises adequate incision and drainage preferably under ether narcosis. Local anaesthesia through novocaine infiltration or ethyl chloride freezing is unsatisfactory. After sterilizing the area with 3.5 per cent tincture of iodine followed by alcohol a T-shaped incision is made as follows. The first incision over the dome of the abscess is made parallel to the external sphincter the second being at a right angle from the center of the first (Fig. 213). After the pus has been evacuated the cavity is sponged

thereby producing a blind internal fistula. Less frequently perforation occurs through the skin near the anus and a complete or blind external fistula follows. When the drainage is insufficient multiple perforations may occur in the ano-perineal skin. The mucosal perforation however is generally solitary. At times a well-developed abscess in one fossa spreads to the opposite side through the posterior cellular space between the sphincters and a *horseshoe fistula* may develop therefrom. Untreated ischiorectal abscesses practically always eventuate in chronic fistulae.

The microorganisms commonly present in ischiorectal abscess parallel those of the intestinal flora. Staphylococci streptococci and *B. coli* predominate. Many anaerobes including the Welch bacillus *Diplococcus reniformis* *Staphylococcus parvulus* and black pigment producing bacilli may also be present. The anaerobic flora account for the gas, foul odor and proteolytic digestion of fat so frequently exhibited.

*Symptomatology* — The symptoms are those of focal suppuration, local pain, chilliness, pyrexia, sweating and leukocytosis. The pain is throbbing in character, worse at night and is accentuated by sitting or defecation. Since the abscess begins in the deeper parts there may be no external evidence of its presence for several days. Ultimately the fossa becomes indurated and the overlying skin red, hot and tender. Early digital examination will often elicit a tender spot and at times a fluctuate bulging. A preexisting history of fissure is seldom obtained in childhood.

*Prognosis* — Early surgical intervention will usually result in cure. Neglected cases almost invariably result in the formation of one or more chronic fistulae.

*Treatment* — This comprises prompt incision and adequate drainage of the abscess cavity. Under general anesthesia and with the patient in the lithotomy position, a wide incision is made from before backward, parallel to and outside of the external sphincter. It is advisable to supplement this by a second incision made at right angles from its center and directed outward, thus forming a T (Fig. 213). This radicle type of incision affords adequate exposure and permits of good drainage.

The abscess cavity should be thoroughly explored with the finger and all necrotic trabeculae broken down in order to convert the multiple pus pockets into one cavity. The latter is sponged dry, swabbed with 95 per cent phenol followed by alcohol and lightly filled with vaseline gauze strips. The error of packing rather than draining the cavity favors sinus formation. The sphincter is then gradually dilated to permit of the passage of gas. The drains are removed on the third or fourth day at which time the cavity is irrigated with Dakin's solution. Daily irrigations and the replace-

ment of vaseline drains are continued until healing occurs. A change of dressings followed by a Sitz bath after each bowel movement is very comforting.

When abscesses are present in both fossæ the parallel portion of the T incision over the more swollen side is prolonged backward to the mid line at the coccyx. A shorter antero-posterior incision is made on the opposite side so as to avoid dividing the posterior raphe.

**Supralelevator Abscesses**—Accumulations of pus in the perirectal space between the peritoneum above and the levator ani below are of rare occurrence. The abscess may be situated either anterior or posterior to the rectum in the superior pelvirectal or retrorectal space respectively or develop in the rectal wall between the mucosa and muscularis.

**Superior Pelvirectal Abscess**—Infection of the anterior space generally occurs through the rectal wall from ulceration or injury by a foreign body. The abscess may rupture into the rectum or burrow upward and either perforate the pelvic peritoneum or point in the inguinal region.

**Symptomatology**—The symptoms are those of a deep-seated abscess. Pain and throbbing in the rectum accompanied by chilliness, pyrexia and leukocytosis. Digital examination usually reveals a tender boggy swelling in the anterior rectal wall.

**Treatment**—This comprises incision and drainage of the abscess through the ischiorectal fossa. Drainage through the rectal wall is inadvisable as a permanent fistula may result.

**Retrorectal Abscess**—Infection may occur from the rectal wall through the lymphatics or the abscess may be secondary to osteomyelitis of the vertebrae or pelvic bones. In the latter instance tuberculosis should be suspected.

**Symptomatology**—The symptoms may vary from a sense of fullness in the rectum to those of severe pain and throbbing. Bogginess or fluctuation of the posterior rectal wall is readily demonstrable by digital examination. Roentgenologic examination of the lower vertebrae and pelvic bones is indicated in obscure cases.

**Treatment**—Incision and drainage of the abscess is preferably performed through the ischiorectal fossa. A crescentic incision between the anus and coccyx is ill advised as the attachment of the sphincter is thereby impaired.

**Interstitial or Mural Abscess**—The abscess usually develops in the lower portion of the rectum in the submucous tissue between the mucosa and muscularis. In rare instances the process pursues a subacute or chronic course.

**Symptomatology**—The symptoms vary from a sense of fullness in the rectum to acute pain and throbbing. Upon digital examination

the boggy swelling is readily palpable, most commonly in the lateral rectal wall. Spontaneous rupture may occur at the pectinate line, followed by the discharge of considerable pus.

*Treatment*—This consists of incision and drainage of the abscess through the rectum. Following dilatation of the sphincter, the mucosa is divided for the full length of the abscess and the pus evacuated. A portion of the mucosal margins is then trimmed away to permit of adequate drainage. After securing hemostasis a No. 20 F. catheter, surrounded by vaseline gauze, is inserted into the rectum well above the wound. The bowels are moved on the fourth postoperative day by administering a milk and molasses enema through the catheter. The latter is usually expelled with the enema and no further treatment is required beyond regulation of the bowels.

## CHAPTER XXXVI

### THE FEMALE GENITALIA

APART from infections the female genitalia of children are little affected by disease. This is especially true if the presentation is limited to surgical entities. It is not until the individual passes puberty that maladies associated with maturation and pregnancy are encountered. For the sake of completeness however the organs constituting the tract will be briefly reviewed.

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#### THE VULVA

Anomalies in structure may vary from total absence to congenital defects. The former condition is often associated with faulty development of the bladder and rectum.

#### ANOMALIES OF THE VULVA

Atresia of the vulva is usually due to adherence of the labia majora and minora as a result of either fetal or postnatal vulvitis with subsequent adhesion formation. If manual separation of the labia is ineffectual the adhesions will have to be incised. Usually a small opening is present in the region of the clitoris through which urine and vaginal secretions pass. The incision may be made with a guide passed through this aperture.

An infantile vulva may persist through adolescence. Underdevelopment of the internal genitalia is usually associated with the condition.

Double vulva is a decided rarity. One observer reported such a case in a woman who gave birth to a child through each opening.

The labia majora and minora may evidence varying degrees of structural change and may be atrophic or hypertrophic. The latter condition is commonly seen as a racial characteristic in the Hottentots.

The clitoris may be rudimentary or hypertrophied. The latter condition is usually associated with hermaphroditic changes and may be congenital or acquired. Surgical intervention is sometimes necessary in the plastic repair for final anatomic sex determination.

Bifid clitoris is chiefly seen in association with extrophy of the bladder and epispadias. Its correction represents a step in the plastic repair of the more important urogenital defect.

## THE HYMEN

### MALFORMATIONS OF THE HYMEN

Malformations are relatively common. The thin fibro-elastic perforated membrane representing a clitoral vestige is subject to many variations in structure. Imperforate hymen is of no consequence in infancy and may pass unnoticed until puberty. The absence of an opening into the vagina in the child is actually a protection against vaginitis.

With the onset of menstruation and the accumulation of menstrual blood behind the impenetrable barrier symptoms evidence themselves either in the form of lower abdominal cramps, tumefaction or urinary difficulties. The entrapped blood may assume such volume as to obstruct the urinary flow by pressure on the urethra or lead to constipation by pressure posteriorly on the rectum. Dysuria, difficulty in urination and even paradoxical incontinence of a chronically distended bladder may occur.

Physical examination of the abdomen may reveal a hypogastric mass representing either a distended bladder or hematocolpos. Inspection of the external genitalia will reveal the tense bulging hymen and rectal examination will confirm the presence of a cystic pelvic mass. Adhesions of the labia minora may lead to a mistaken diagnosis.

**Treatment**—Incision of the hymen generally results in the immediate relief of symptoms. In some cases however the long standing obstruction to urinary outflow produces the picture of vesical neck obstruction characterized by distended bladder, dilated ureters and a varying degree of hydronephrosis either with or without superimposed urinary tract infection. Treatment of such conditions must be continued following the incision of the hymen. (Refer to the chapter on Urinary Obstruction.)

**Absence of the Hymen** hardly ever occurs and in suspected cases careful inspection of the area will usually reveal some remnants of the fibromuscular membrane. The form of the hymen may be that of a membrane with numerous openings, the cribriform type or it may be circular or eccentric or divided into two halves as a septate membrane. Such findings are of little concern unless an acute or persistent vaginitis assumes such severity as to make more adequate drainage necessary. In such an event incision and the creation of an adequate channel is indicated.



inflammatory retention. In certain instances, debility may account for vaginal discharge.

**Symptomatology** —The symptoms include swelling and redness of the labia, vestibule and vagina. The discharge may be a thin, watery mucoid material, or it may be creamy thick in consistency. Extension of the infection to the urethra will cause dysuria and frequency of micturition, while failure to keep the area clean will produce varying degrees of skin excoriation.

**Diagnosis** —Diagnosis lies in microscopic examination of the secretion and in the clinical course. The non-specific type has none of the extreme contagiousness of the gonorrheal type. The latter tends to extend into the vagina and cervix while the former type is generally localized to the vulva.

**Treatment** —Treatment is directed at removal of the cause. The parts may be kept clean by frequent lavage with 1 to 5000 potassium permanganate solution or 1 to 10 000 bichloride of mercury. A 5 per cent solution of freshly prepared argyrol may be introduced into the vagina through a small catheter passed through the hymeneal aperture. The surrounding skin may be protected by a layer of boric or zinc oxide ointment.

**Herpetic Vulvitis** —Herpetic vulvitis, analogous to aphthous stomatitis, may occur in the exanthemata notably varicella. Secondary infection may be engrafted on these sites of lowered mucosal resistance.

**Diphtheritic Vulvo-vaginitis** —Diphtheritic vulvo-vaginitis may occur either with or without a similar infection of the nasopharynx. A grayish or yellow membrane may cover the red edematous mucous membranes of the vulva and vagina. Treatment comprises the administration of diphtheria antitoxin and local irrigations with antiseptics.

**Abscess of the Vulva** —Abscess of the vulva, as a result of trauma or as a sequela of severe infection, may demand incision and drainage if the conservative application of wet dressings is ineffectual.

**Erysipelas of the Vulva** —Erysipels of the vulva occurs in the debilitated or neglected child. It manifests itself by redness and edema of the labia, burning, and evidence of systemic toxemia. Treatment is supportive with local therapy to keep the parts clean. Recently heavy doses of ultra-violet light have been used effectively to check erysipelas. Serum also has its proponents and very recently synthetic antistreptococcus dyes for oral subcutaneous and intravenous use have been tried. Final acceptance must be deferred until sufficient clinical evidence of the effectiveness is obtained. At times the infection may be extremely virulent with rapid extension through the vagina, the uterine cavity and Fallopian tubes into the peritoneal cavity with resulting fatal peritonitis.

**Complications** — These are relatively infrequent and may include inguinal adenitis with or without suppuration or bubo formation urethritis or pyelonephritis proctitis salpingitis or pelvic peritonitis gonorrheal ophthalmia arthritis endocarditis or meningitis Harris and Beriman (1934) recorded 34 cases of gonorrheal peritonitis in young girls in addition to 2 of their own. A 50 per cent mortality attended operative measures as compared to 24 per cent with conservative therapy (the latter appears especially high).

**Treatment** — The therapy of gonorrheal vulvo-vaginitis should be twofold. A rigid prophylactic regime will minimize the spread of the infection in hospitals, schools and homes. Routine vaginal smears on admission and weekly examinations thereafter are practised in well organized institutions caring for children. In private practice every vaginal discharge should be examined and the parents carefully instructed in the care of clothes, utensils and possible contacts with other children. Whoever is entrusted with the care of children, mother, nurse or teacher, should be instructed in the nature, course and significance of the disease for only in this way can epidemics be avoided.

General therapy follows along lines similar to those already laid down for non specific vaginitis. Specific measures include twice-daily douches of 1 to 5000 potassium permanganate or 1 to 10 000 bichloride of mercury solution using a small catheter for vaginal irrigation. Following this 10 per cent argyrol, 5 per cent neosilol, 1 per cent protargol or 1 per cent mercurochrome solution is introduced into the vagina and a protective vulva pad applied. The instillation is best carried out with the child lying on her back, hips elevated and thighs flexed acutely on the trunk. This position not only facilitates administration of the drug but keeps it in contact with the greatest surface area for the longest time. The drugs that have been used are legion in the form of solutions, suppositories or jellies.

Fever therapy produced by foreign protein injections (milk, adjuvant typhoid) or by artificial means (baths, thermal boxes) have been used to distinct advantage by some. The rationale of the procedure lies in the optimum bacteriostatic and bacteriocidal temperatures for the gonococcus.

Vaccine therapy using filtrates of the gonococcus has been lauded by some while others see little value in its use. Lewis (1933) suggested the use of estrogenic substance in the form of theelin and reported good results. Numerous reports have subsequently been made on this and other female sex hormones, some laudatory, others frankly derogatory. The rationale for such endocrine therapy lies in the fact that hormonal elaboration by the maturing ovarian follicle produces vaginal epithelial proliferation. The delicate cells

with deeply staining nuclei are converted into hardy stratified squamous epithelium

The ovarian follicular hormone (theelin, amniotin) may be given orally subcutaneously or in the form of vaginal suppositories. Dosage, calculated in rat units, must be adjusted to the individual patient's response to therapy as noted clinically and through the medium of microscopic studies of vaginal cytology and bacteriology.

The subject of gonorrheal vulvo-vaginitis is a complex one and its clarification remains a problem of the future. Benson and Steer (1937) have reviewed the extensive literature and note considerable confusion in reported observations on the efficacy of any particular therapy and the criteria of cure.

Treatment should be carried on beyond the date of cessation of vaginal discharge. Under no circumstances should the child be discharged as cured unless she has been free of vaginal discharge for at least three months with repeatedly negative smears. The disease is characterized by remissions and exacerbations and this fact should guard the physician in judging the value of any particular therapeutic agent as well as the permanency of cure.

### TUMORS OF THE VULVA AND VAGINA

New growths of the vulva and vagina are rare. Warty excrescences are readily amenable to cauterization either with chemical caustics such as silver nitrate or trichloroacetic acid, or the spark-gap electrode.

Vaginal Polyps are occasionally found in children. Their discovery usually follows upon the investigation of vaginal bleeding. The polyp may be removed, depending upon its accessibility, with a snare or by applying a loop ligature to its base and either amputating the polypoid mass or allowing it to slough off.

Benign Tumors that have been reported include cysts, fibromata and myomata. Malignant tumors are fortunately rare. Isolated cases of sarcoma or carcinoma have been reported and sarcoma of the vagina apparently occurs more often in children under five years than in adults. Mergelsberg (1913) collected 37 cases of malignancy in children from the literature. Vaginal bleeding and the rapidity of extension to adjacent tissues characterize their presence. Ulceration is a prominent symptom while cachexia appears late in the disease. Chorioepithelioma has been seen in a few instances in girls at or about puberty. When recognized the condition is usually too late for any surgical intervention.

## THE UTERUS

Anomalies of the uterus are frequent especially in association with maldevelopment in other structures of the genital or urologic tract. The organ may be absent rudimentary infantile or maldeveloped.

### ANOMALIES OF THE UTERUS

**Uterus Unicornus** represents an organ in which a single horn has developed while the *bicornuate* uterus represents a two horned organ in which the duct segments have failed to fuse. The double uterus or *uterus didelphys* represents the extreme of this condition. Rarely is an accessory uterus discovered. All of the above anomalies are merely of academic interest since their presence is noted only at the time of laparotomy or at postmortem. Disturbances in menstruation call the pediatrician's attention to the possibility of their existence.

**Prolapse of the Uterus** has been noted in the new born and in early infancy. Spina bifida a concomitant finding in over 80 per cent of the cases has been considered a causal factor on the basis of disturbed innervation of the pelvic supporting musculature. Other possible etiologic factors include malnutrition visceroptosis congenital widening of the genital hiatus and oversized pelvis. Treatment is conservative until the child is older. Tampons or packs may be used for support.

**Hematometra** results from the retention of menstrual products over a prolonged period of time due to atresia of the vagina or an imperforate hymen. Removal of the cause results in cure.

### INFECTIONS OF THE UTERUS

Primary infections of the uterus are rare. Secondary involvement may follow from an infective pyogenic or tuberculous process in the pelvis or Fallopian tubes. Ascending infections from the vagina may involve the uterine cavity. The latter is relatively resistant to the gonococcus. (Refer to *Gonococcus Peritonitis*.)

### TUMORS OF THE UTERUS

Tumors of the uterus are uncommon. Fibromyomata are seldom seen even at the autopsy table. The malignant growths are chiefly sarcomatous. McLain (1927) found 12 reported cases in the literature several with involvement of both uterus and vagina. Like other juvenile malignancies these tumors are usually beyond surgical intervention. Deep roentgen ray therapy in radio-sensitive growths is effective in temporarily reducing their size as well as in arresting hemorrhage.

## THE FALLOPIAN TUBES

Varying degrees of malformation may occur. One or both tubes may be absent or be represented by a fibrous cord or they may be extremely long and tortuous. Such anomalies together with malformations of the infundibular end represent etiologic factors in later sterility and ectopic gestation. The incidence of tubal infection in early life is comparatively uncommon. Tuberculous salpingitis is almost always secondary to intestinal or peritoneal involvement. Gonococcal infection is usually localized to the vulva and vagina and salpingitis or pyosalpinx similar to the adult type seldom eventuates. (Fig. 179)

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## THE OVARIES

Anomalies of the organs may be represented in total absence atrophy hypertrophy supernumerary or congenital displacement. Displacement assumes significance when the ovary is included in an inguinal or femoral hernia. Twisting and ultimate strangulation of the ectopic ovary may occur exhibited by pain swelling and systemic reaction. Reduction of the displacement generally results in relief of symptoms. This may be carried out by digital manipulation or at the time of operative repair of the hernia.

Aberrant ovarian tissue has been found attached to peritoneum omentum and intestine. Such tissue is said to be more susceptible to malignancy and also accounts for the rare cases of pregnancy or continued menstruation after bilateral oophorectomy has been performed.

## OVARIAN TUMORS

Cystic and solid tumors of the ovary are among the more common abdominal growths of childhood. Approximately one-half are simple cystomas or dermoids while the remainder are teratoma sarcoma or rarely carcinoma.

Although the term dermoid designates a tumor composed solely of epiblastic tissue no ovarian tumor containing only ectodermic elements has ever been described. Through common usage the term is applied to a large class of cystic tumors in which epiblastic derivatives predominate and the malignancy potential is exceedingly slight. When the tumor contains a large proportion of embryonal cells and for this reason is peculiarly prone to malignancy it is called teratoma.

**Etiology** The origin of these tumors has aroused much controversy. Waldeyer and Wiens contend they are ovigenic. Cohnheim that they arise from early ectodermal inclusions and Kromer that

they are of ovarian origin the cystic element coming from the follicle and the tissue elements from the ovule. The cystic types (dermoids) are more common and are seldom subject to malignancy (Ewing estimates the rate at 3 per cent.)

**Symptomatology**—The tumors may be congenital or occur at any age and are occasionally bilateral. Swelling in the lower abdomen generally attracts attention to the condition (Fig 214). In some instances however pain from torsion of the pedicle is the first symptom. Such cases are frequently mistaken for acute appendicitis. Simple cysts may attain tremendous size and even cause respiratory embarrassment. Malignant tumors are less subject to massive growth and often remain asymptomatic until progressive anemia and loss of weight occur. In older children an ovarian tumor may produce signs of precocious sexual development.

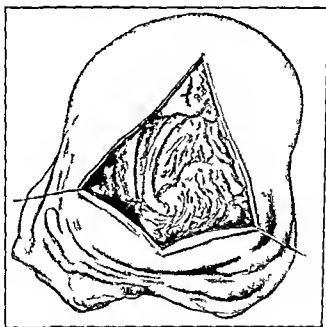


FIG 214—Dermoid cyst removed from a child aged two years eight months

**Diagnosis**—The diagnosis at times is puzzling. A cyst with a long pedicle may lie entirely above the pelvis and be mistaken for a mesenteric cyst or hydronephrosis. Bimanual examination with one finger in the rectum is often helpful in the differential diagnosis. Huge cysts may simulate ascites or tuberculous peritonitis. In the presence of free fluid the summit of the abdomen is tympanic whereas in cystic accumulations it is flat. Roentgen examination after an opaque meal will often determine the relationship of the mass to the viscera. Urography will assist in the differentiation

from renal masses. A cystoma with torsion of the pedicle may mimic acute appendicitis. The presence of a mass immediately following the onset of symptoms excludes the appendix.

**Treatment**—Cystectomy is indicated for benign tumors. In operating for acute appendicitis in girls the pelvis should always be explored for the possibility of a cyst if the appendix is found normal. The therapy of malignant growths chiefly sarcoma, is discouraging because recognition of the condition occurs at a late stage. Radiation as a pre- and postoperative measure should be considered. Extirpation of the growth is indicated whenever possible.

### VAGINAL BLEEDING

Flow of blood from the vagina in childhood should demand early and thorough investigation. It may result from inflammatory ulceration of the vulva, vagina or cervix or follow local trauma. It may also be the first sign of an uterine or ovarian tumor, or be due to a hemorrhagic diathesis or occur without any evident pathology. Such instances are usually referred to as precocious menstruation with or without other evidences of sexual maldevelopment. Ovarian or adrenal tumors should be sought.

The treatment of genital hemorrhage depends upon the cause of the condition.

## CHAPTER XXXVII

### HERNIA

HERNIA is a common condition in infancy and childhood. An incidence of 0.8 per cent was reported by Patterson and Gray (1927) who reviewed the case histories of 130-243 children under five years of age. The following varieties merit special emphasis: (1) Umbilical (2) inguinal (3) incisional (4) femoral and (5) diaphragmatic. Rare forms will be only briefly discussed.

**Historical**—The term rupture has probably descended from the time of Galen who taught that hernia was due to a rupture of the peritoneum. For centuries various bandage supports were worn. Gordon (1306) employed an iron truss and Le Quin (1678) introduced the steel type. During the eighteenth century Littre, Petit, Gimbernat, Camper and Richter revived interest in hernia and contributed valuable anatomic knowledge.

Modern therapy dates from 1880 when free exposure and plastic repair were advocated. Bassini published his classical inguinal herniotomy in 1888 and a year later Halsted advocated extra-aponeurotic transplantation of the cord. Wolfier (1892) incised the rectus sheath and anastomosed the muscle to Poupart's ligament. Andrews (1898) recommended imbrication of the transversalis aponeurosis and Bloodgood (1898) advocated transplantation of the rectus muscle in certain types.

Herniotomy without transplantation of the cord was first advocated by Coley (1895) and Ferguson (1899). This is the present day operation of choice for indirect hernia in children. It is also indicated when the condition is associated with maldescent of the testis.

Fascial suture was suggested by MacArthur (1901) who employed a strip of the external oblique aponeurosis. The real value of living fascial suture for repair was further emphasized by Gallie and Le Mesurier (1921) who recommended that strips be taken from the fascia lata. Although this technic with certain modifications is of special value in the treatment of large direct and recurrent hernie, it is rarely indicated in children.

Certain cures following the injection of astringents have recently been published. Such procedures are potentially dangerous and are not recommended.



*A Anatomic Classification*

- 1 Umbilical
- 2 Inguinal
- 3 Incisional postoperative or ventral
- 4 Femoral
- 5 Epigastric
- 6 Miscellaneous rare forms including
  - (a) Diaphragmatic
  - (b) Lateral ventral
  - (c) Lumbar
  - (d) Obturator
  - (e) Sciatic
  - (f) Perineal
  - (g) Pudendal
- 7 Internal hernia

The foregoing are arranged in order of their incidence in early life

*B According to Contents*

- 1 Omental hernia
- 2 Intestinal hernia or enterocele
- 3 Hernia of the bladder
- 4 Sliding hernia of the colon

Although every abdominal organ except the pancreas and liver has been found in some form of hernia the foregoing comprise the usual varieties

*C Clinical Types*

- 1 Reducible hernia
- 2 Irreducible hernia
  - Complications of irreducible hernia
    - (a) Obstructed hernia
    - (b) Inflamed hernia
    - (c) Strangulated hernia

**CLINICAL TYPES OF HERNIA**

**1 Reducible Hernia**—This term is applied to all types of herniæ whose contents are replaceable within the abdomen either by posture or light taxis. The vast majority of herniæ in childhood are of this type.

**2 Irreducible Hernia**—Although the hernial contents are not replaceable the term implies the preservation of normal function and vascular competency of the bowel. The irreducibility is generally caused by omental adhesions to the sac wall rather than narrowness of the hernial aperture. Sliding hernia is always partially irreducible.

**(a) Obstructed Hernia**—The obstruction to the passage of feces and gas results from fecal impaction without circulatory interference.

Although of frequent occurrence in elderly patients with irreducible hernia, the complication rarely develops in early life

*Symptomatology* — The onset is gradual with progressive constipation eventuating in obstruction so that neither feces nor gas is passed. Abdominal distention follows but nausea and vomiting are usually late symptoms. The obstructed hernial mass is not tender or only slightly so. The development of acute tenderness indicates inflammation or strangulation and demands immediate operation.

*Treatment* — Repeated enemata generally relieve the obstruction. Taxis and cathartics are dangerous. Obstinate obstruction usually denotes circulatory interference and prompt surgery becomes imperative.

An obstructed hernia is sometimes referred to as an "incarcerated hernia." The latter term is loosely applied to any complication of hernia varying from irreducibility to strangulation. This lack of specific definition should preclude its usage.

(b) *Inflamed Hernia* — The condition ensues when the contents become inflamed. The pathology is in reality a localized peritonitis and the process may be acute or extremely mild. The former is unusual in childhood and the inflammation is generally slight and unaccompanied by focal symptoms of pain or tenderness. The occasional presence of omental adhesions is an evidence of previous mild inflammation without symptoms.

Inflammation of the sac contents results at times from trauma or an ill-fitting truss and in rare instances from enteritis, tuberculosis or appendicitis. The symptoms of focal pain and tenderness depend upon the acuity of the process.

*Treatment* — This comprises rest in bed and the application of an ice-bag to the parts. Unless the inflammation subsides promptly, operation should not be delayed. *the differential diagnosis of inflamed hernia from beginning strangulation is a dangerous refinement*

(c) *Strangulated Hernia* — Circulatory damage is the basic factor in strangulation. Although the vascular incompetency may concern only the omentum, the term "strangulated hernia," through common usage, implies intestinal damage. The latter seldom occurs in children because the surrounding structures are soft and elastic.

*Pathology* — The dominant elements are vascular thrombosis and tissue necrosis. When confined to the omentum, the latter becomes dark in color, tensely edematous and surrounded by turbid exudate, the end result being a local peritonitis of the sac. The pathology of strangulated intestine is described under Intestinal Obstruction.

*Symptomatology* — It should be emphasized that an anatomic and pathologic diagnosis between inflamed, obstructed and strangulated hernia is frequently impossible. Any hernia which suddenly becomes

larger and irreducible tense and tender and which is accompanied by abdominal pain nausea and vomiting should be considered strangulated and imperatively operable. The same rule applies to all cases in which there is the slightest doubt as to vascular competency. Procrastination is inexcusable and extremely dangerous.

**Treatment**—This comprises immediate operation. When the omentum is involved the structure should be sufficiently withdrawn to inspect the line of demarcation. The thrombotic area is best resected at least 1 cm. proximal to the zone of strangulation and the stump ligated with plain cat gut chain sutures. Meticulous hemostasis is imperative as omental bleeding is unfavorable to clotting. The treatment of strangulated intestine is described under Intestinal Obstruction.

### UMBILICAL HERNIA

Hernia at the umbilicus occurs frequently in early life. The cases may be divided into three groups: (1) Hernia into the umbilical cord; (2) infantile or acquired hernia; and (3) adult umbilical hernia. The first two are admittedly due to congenital developmental defects and the adult type being anatomically identical with the infantile also strongly suggests a congenital origin.

**Development of the Umbilicus**—During early fetal development the lower ileum and cecum are contained within the cord outside of the abdominal cavity, being later withdrawn into the abdomen. The peritoneal covering extending into the cord is also retracted and at birth presents as a slight depression at the umbilical ring. Defective withdrawal into the abdominal cavity of either the viscera or peritoneum eventuates in hernia into the cord.

After ligation of the cord the umbilical ring closes as follows. The stump covered by amnion dries and sloughs off and the granulating area becomes epithelialized. The superficial fascia transversalis and peritoneum fuse with the skin and produce the thin cicatrix which normally covers the umbilical ring. At the latter site the rectus sheaths do not quite come into contact. Fibrous obliteration of the hypogastric arteries and urachus strengthens the lower half of the ring and tends to pucker the umbilicus inward, whereas obliteration of the large umbilical vein leaves a definite weakened area in the upper portion. It is through this hiatus formerly occupied by the umbilical vein that infantile and adult types of umbilical hernia evaginate, the rounded protrusion appearing above the cicatricial depression.

**The Sac**—The hernial sac of peritoneum is fused with the attenuated fibers of the transversalis and superficial fascia. Through absence of subcutaneous fat the overlying skin is also intimately associated with the sac and forms its main covering. Thus at

operation, the thin wall appears to consist only of skin and peritoneum. The contents are commonly omentum, occasionally a loop of small intestine, and infrequently a portion of the transverse colon.

### Clinical Varieties of Umbilical Hernia.

**Hernia Into the Cord.**—This type, also termed *congenital hernia*, is fortunately rare, the incidence ratio being about 1 to 5184 births (Landfors). The rope-like umbilical cord which normally emerges from a small navel opening is replaced by a funnel-shaped defect through which viscera may protrude into the cord, clearly visible within the greenish-white transparent amniotic covering.



FIG. 215.—Eventration

The hernia may vary in size from a small bulging at the base of the cord to complete eventration. Every umbilical cord which is swollen at the navel attachment should be carefully examined lest its ligation include intestine; strangulation produced thereby will result in death or fecal fistula. The hernia generally contains coils of small gut but in extreme types practically every abdominal organ except the pancreas may participate in the eventration. (Fig. 215) Spontaneous rupture of the sac with resulting evisceration may occur during delivery or from subsequent infection. The infants are frequently premature and often exhibit other abnormalities.

**Prognosis.**—The outcome depends chiefly upon the degree of herniation and the promptness of surgical intervention. Many cases of moderate size recover if operation is performed within a

few hours of birth. Extreme cases of eventration which are not subject to repair rarely survive. Death generally occurs within ten days from exsiccation, necrosis, peritonitis or sepsis.

**Treatment** — After the umbilical cord has been ligated and amputated distal to the sac, the infant should be wrapped in a sterile sheet and be referred to the surgeon for immediate operation. Spontaneous recovery following ligation occurs only in exceptional cases of very small herniation. Delay is dangerous from the standpoint of both infection and necrosis of the sac. The average type is reducible and subject to herniotomy; repair by the method described under the treatment of Infantile Hernia. In extreme cases, plastic closure may be impossible through failure of development of the abdominal muscles.

**Infantile Hernia (Acquired Umbilical Hernia)** This common but seldom serious affection develops generally in the first few months of life and rarely after the third year. The incidence is highest in delicate infants and in those subject to flatulence, excessive crying, straining efforts from constipation or phimosis and other conditions which increase intra abdominal pressure.

**Symptomatology** — Herniation occurs through the hiatus in the upper portion of the navel at the site of the obliterated umbilical vein. The tumefaction appears as a small elastic non sensitive swelling covered by skin and varies in size from a slight convex bulge to that of a marble, being globular or slightly irregular in shape. The contents consist of intestine which is almost always readily reducible. Inflammation and strangulation are rarely observed.

**Prognosis** — Spontaneous cure will occur in most cases without any form of treatment from progressive closure of the ring through development of the recti muscles. Recovery may be hastened however by appropriate treatment.

**Treatment** — Prophylaxis is important. Infants subject to abdominal distention or persistent straining efforts should wear a firm umbilical compress under an abdominal band for the first few months. If a hernia is present it should be kept constantly reduced until the umbilical opening has had time to close. A common procedure consists in applying a 2 inch band of adhesive plaster with a chamois covered coin so placed that pressure will be exerted over the navel. The dressing should be worn continuously and be changed only when necessary. At such times boric acid powder may be applied to the navel. Should the skin become irritated a tight fitting cotton flannel binder may be substituted. Appliances with a rounded surface should never be employed since they push the hernia inward and thereby keep the umbilical opening dilated. A cure usually obtains in three to six months.

Palliative treatment is often unsatisfactory in infants past the first year. The parents should be advised that the condition is

rarely dangerous and almost always disappears with development. Surgery only becomes indicated when the hernia enlarges or persists after the tenth year and in rare instances of irreducibility or strangulation.

### INGUINAL HERNIA

This is the commonest form of hernia except during the first few weeks of life when the umbilical predominates. Although the condition may occur in three different forms practically all cases are of the oblique or indirect type, direct hernia and the combined direct indirect being exceedingly rare.

**Indirect Inguinal Hernia (*Oblique Inguinal Hernia*)**—In indirect hernia the sac emerges through the internal abdominal ring lateral to the deep epigastric vessels and passes obliquely downward and inward along the inguinal canal to emerge through the external abdominal ring. The condition may be congenital or acquired. Depending upon the length and position of the sac the following types are recognized:

1 **Scrotal or Congenital Hernia of the Tunica Vaginalis**—The funicular process of the peritoneum is completely patent and the intestine descends to and at times surrounds the testis.

2 **Complete or Funicular Hernia**.—The contents pass out through the external ring a variable distance into the open funicular process at times down to but not communicating with the closed tunica vaginalis of the testis.

3 **Incomplete Hernia or Bubonocoele**—The hernia lies entirely within the inguinal canal and does not pass beyond the external ring due to closure of the funicular lumen at some point within the canal.

**Incidence**—According to Keith 4.4 per cent of male infants have oblique inguinal hernia in the first year of life and of these approximately two-thirds are cured during childhood either spontaneously or through the aid of a truss. This percentage of cures accords with 1.7 per cent incidence of inguinal hernia in adults as recorded by the Drift Board during the World War. The right side is affected in 60 per cent of cases such predominance being due to the later descent of the right testis and closure of the right funicular process. The sex ratio is approximately 10 males to 1 female. In about one-fourth of the cases the condition is bilateral.

**Etiology**—Persistent patency of the funicular process or of the canal of Nuck is the dominant predisposing factor in the development of indirect inguinal hernia. This congenital defect of closure obtains in approximately 30 per cent of infants up to four months after birth and persists throughout life in about 10 per cent.

**Congenital Saccular Patentcies**—These are similar in both sexes and the ratio of 10 males to 1 female results from both anatomic

and mechanical differences. Due to testicular descent the male funicular process is larger and longer and the internal and external rings are increased in size. Moreover opposing forces obtain in the two sexes. Whereas the size, weight and mobility of the testis exerts an outward drag upon the funicular process, the female sac is retracted inward through the uterine pull upon the round ligament.

*Intra-abdominal Pressure* — This is the most important exciting factor in forcing visceral contents into the preformed peritoneal sac with resultant hernial formation. Keith and others reject the saccular theory and stress the importance of strain as the chief causative factor. They maintain that the involuntary contraction of the conjoined internal oblique and transversalis fibers against Poupart's ligament acts as a shutter buffer to close the potentially weak inguinal region.

The writer has been impressed by an anatomic arrangement which frequently occurs in oblique herniæ of children: shortness and straightness of the canal, increased obliquity of Poupart's ligament and the relative transverse direction and frailty of the marginal fibers of the conjoined muscles. Such architectural arrangement conduces to definite weakness. On numerous occasions the same muscular arrangement has been demonstrated on the opposite side although a hernia was not manifested clinically. Careful dissection of the internal ring in such cases has always disclosed a peritoneal protrusion of at least 1 cm. thus evidencing a potential bubonocoele.

A preformed sac from persistent patency of the funicular process plus the element of increased abdominal pressure incident to strain are generally accepted as the chief factors in the production of indirect inguinal hernia. In certain instances muscular incompetency may be an associated element. Depending upon the length and character of the patent processus vaginalis the hernia may first appear as a bubonocoele, small complete hernia or large scrotal type. Cases occurring at birth or soon thereafter are usually due to congenital patency of the entire funicular process and often extend into the scrotum. Those developing later probably begin as a bubonocoele and gradually extend beyond the external ring due to repeated strains from various causes such as abdominal distention, excessive crying or coughing, effort at stool from diarrhea or constipation, straining on urination from phimosis or gravel and in later childhood from vigorous playing. The majority of cases develop during the first year.

*Symptomatology* — The hernia is situated more often on the right side and is bilateral in approximately 25 per cent of cases. Subjective symptoms are usually wanting. The swelling in the inguinal region imparts an expansile impulse on coughing or straining and is generally readily reducible by either posture or light taxis. Although large herniæ occur quite commonly in male infants the

bubonocoele and small funicular types predominate in girls, large labial types are rarely observed

The hernial content generally consists of small intestine with or without omentum. In unusual cases the cecum, appendix, Meckel's diverticulum, sigmoid, iliocecal tube, ovary or bladder may occupy the sac.

**Diagnosis.** The chief difficulty in diagnosis is in distinguishing hernia from *hydrocele*. The latter is tense, irreducible, transilluminates light, has no expansile cough impulse, and the spermatic vessels are palpable above the upper pole. At times the two conditions are associated. *Hydrocele of the cord* may produce a small oval swelling in the canal simulating bubonocoele; the former is tense, elastic, irreducible and exhibits no expansile impulse. *Femoral hernia* is rare. Its presence is indicated if the hernia reappears while the tip of the finger obstructs the external ring. *Inguinal adenitis* and *lipoma* offer little difficulty in differentiation; the former is nodular and often tender and the latter soft, superficial and lobulated; expansile cough impulse and reducibility are wanting in both. *Psoas abscess* may be confused with hernia; a spinal roentgenogram will readily indicate the pathology.

**Prognosis.**—Approximately one-third of the cases require herniotomy. Small herniae often disappear spontaneously; average cases are at times completely and permanently cured by the application of a suitable truss; large congenital types and those developing in late childhood are least subject to recovery through palliative support. Strangulation is a rare complication and occurs most often in the first two years and generally before the third month.

**Surgical Anatomy.**—Only structures of surgical importance will be considered as the inguinal canal is fully described in text books on anatomy.

**The External Ring.**—The hiatus between the inner and outer pillars of the external oblique normally measures less than 0.5 cm. in diameter in early childhood and will not admit the tip of the little finger. The intercolumellar fascia sweeping across it is frail and poorly developed.

**Internal Ring.** This is situated midway between the anterior iliac and pubic spines and is a small oval opening in the transversalis fascia bounded above and laterally by the arching fibers of the internal oblique and transversalis muscles, medially by the deep epigastric vessels and below by Poupart's ligament.

**Conjoined Tendon.**—The structure is composed of the marginal fibers of the internal oblique and transversalis muscles which blend into a tendinous layer and stretch across the inner two-thirds of Hasselbach's triangle. They are inserted into the pubic crest and inner part of the ileopectineal line behind the external ring thus protecting an otherwise weak area in the abdominal wall. This



arrangement is adequately supportive in early life and direct hernia rarely develops when present the conjoined tendon is either attenuated or unrecognizable

*Inguinal Canal* —The canal varies in length from approximately 1 cm. at birth to 4 cm. in late childhood. In early life it is relatively straight and as the child develops the canal becomes directed downward and inward toward the pubic spine. It is bounded in front by the aponeurosis of the external oblique and at the lower end by the marginal fibers of the internal oblique above by the arching fibers of the internal oblique and transversalis muscles below by the shelving border of Poupart's ligament and by Gimbernat's ligament and the floor or posterior wall is formed by the transversalis fascia conjoined tendon and triangular ligament.

*Cremasteric Muscle* —This muscle is relatively frail and underdeveloped before puberty. Its fibers surround the cord and extend from the lower margin of the internal oblique into the scrotum. Unless the fibers are meticulously dissected from the hernial sac troublesome oozing may occur. When suturing the conjoined muscles to Poupart's ligament the cremasteric fibers and fascia should not be included as their structure is unfavorable to firm musculo-aponeurotic healing. There is no necessity for resecting the fibers however as they may be readily tucked well upward with the cord away from the suture line.

*Spermatic Cord* —This consists of the vas deferens and the spermatic artery and veins the latter constituting the pampiniform plexus. When dissection of the sac is carefully carried upward to the internal ring the structural arrangement is as follows: the neck of the sac is placed above and between the cord elements the vas deferens diverging medially toward the pelvis and the spermatic vessels backward and upward. In the inguinal canal the sac generally lies antero-medial to the cord.

*Vas Deferens* —In early life the vas deferens is small and delicate and resembles a white linen thread in size and color. It possesses its own minute nutrient artery and whether the vessel itself is sufficient for testicular blood supply remains controversial. When operating for undescended testis the author has occasionally divided the spermatic vessels through necessity leaving only the artery to the vas for testicular nourishment. In no known instance has atrophy occurred or gonadal development been impaired.

*Poupart's Ligament* —Developed from the lower border of the aponeurosis of the external oblique the ligament extends from the iliac spine to that of the pubis and thence along the ileopectineal line continuous with Gimbernat's ligament. While the conjoined muscles are being united to it the external iliac vessels may be protected from injury by lifting the shelving border upward with thumb forceps before inserting the sutures.

**Nerves** —The genital branch of the Genitocrural Nerve lies behind the spermatic cord the inguinal branch of the Ileoinguinal accompanies the cremasteric muscles and emerges through the outer ring and the hypogastric branch of the Ileo-hypogastric lies upon the internal oblique above its lower border in close association with the aponeurosis of the external oblique and pierces the latter a little above and to the outer side of the external ring Sympathetic fibers accompany the blood vessels

The ileoinguinal and ileohypogastric nerves are most liable to damage in herniotomy Injury to the former may result at times in testicular neuralgia damage to the latter apparently affects only its sensory function as all the motor fibers to the internal oblique muscle are given off before the nerve enters the inguinal canal

Emerging from the internal ring the cord receives a covering from the transversalis fascia termed the infundibuliform fascia and then becomes surrounded by the cremasteric muscle and its fascia The sac of an indirect hernia has similar coverings and is intimately associated with the vas deferens and spermatic vessels

### **Uncommon Types of Indirect Inguinal Hernia**

**1 Encysted or Infantile Hernia** —This is a rare form in which obliteration of the funicular process has taken place only at the internal ring the remaining portion being patent With the development of hernia the sac may either evaginate into or pass behind the funicular process A double layer of peritoneum may thus be presented at operation

**2 Interstitial Hernia** In this rare type the sac lies between the tissue planes in the inguinal region The most common aberrant site is between the external oblique aponeurosis and the internal oblique muscle Maldescent of the testis may be an associated condition when the sac is of the congenital type

**3 Hernia Associated With Hydrocele** —This occurs uncommonly in children In its embryonic descent into the scrotum the testis is accompanied by the vaginal process of the peritoneum Prior to birth or soon thereafter the latter becomes obliterated at the internal ring and also just above the testis The lower open portion forms the tunica vaginalis and between the closure points the process becomes a fibrous strand Closure only at the suprastesticular site may result in hernia with hydrocele Hydrocele of the cord develops more commonly however from persistence of the funicular process after closure of its ends The condition may present as a solitary elongated cystic mass or a multiple rosary bead tumefaction

**4 Hernia Associated With Mal descended Testis** —The condition is discussed in Chapter XLIII

**Sac Contents.**—The sac of an inguinal hernia generally contains omentum (epiplocele) or ileum (enterocele), or both structures, and infrequently the cecum, appendix, Meckel's diverticulum, sigmoid, bladder or Fallopian tube and ovary

**Direct Inguinal Hernia**—In direct hernia the sac evaginates mesial to the deep epigastric vessels through Hasselbach's triangle and either penetrates through, or pushes ahead of it, the transversalis fascia and conjoint tendon. Although approximately one-fourth of adult inguinal herniæ are of the direct type, the condition is extremely rare in early life. In male children the incidence ratio of direct to indirect hernia is less than 0.2 per cent and in females the direct type is an anatomic curiosity.

**Diagnosis**—The globular shaped sac is situated at the lower end of the inguinal canal, close to the rectus muscle, and does not tend to enter the scrotum. The hernia is readily reducible and the examining finger passes directly backward through the hiatus rather than upward and outward as occurs in indirect hernia. Although the epigastric vessels cannot be palpated, the diagnosis is suggested by the globular shape, wide mouth and low position of the sac.

**Direct-Indirect Inguinal Hernia**—As the name implies, direct-indirect hernia is a combination of both varieties in which the 'saddle bag' or 'pantaloon' sac is composed of two components, one lying lateral and the other mesial to the deep epigastric vessels. The condition is exceedingly rare in children.

### Treatment of Inguinal Hernia

Palliative supportive treatment is always indicated in infants. The majority of the cases will recover completely and permanently under the application of a suitable truss and the removal of any contributory cause. The support should be worn constantly day and night, and the mother should be impressed with the importance of never allowing the hernia to descend. Should this occur, the truss must never be reapplied until the hernia has been reduced. The appliance is best worn during the bath, being only temporarily removed for cleansing the skin and applying borated talcum.

Through constant mechanical support many herniæ in young infants are cured within three to six months. The truss should be worn for an additional year, however, to prevent possible recurrence. Although bubonocèles and small herniæ may disappear spontaneously, the aid of a truss hastens and insures recovery. Large scrotal herniæ are least subject to cure by palliative support.

In early life a truss may be worn without difficulty or apparent discomfort. In older children, however, it is often impossible to keep the appliance from becoming displaced during periods of vigorous activity. Under such conditions the truss should be

abandoned lest it be replaced after the hernia has descended and produce inflammation of the contents.

**Indications for Herniotomy**—Operation is advisable in all cases which are not benefited by mechanical support after a reasonable trial of a year or perhaps longer in older children who are more or less unmanageable and when the condition is complicated by mal-descent of the testis hydrocele of the cord or irreducibility. In flamed and strangulated herniae demand immediate surgery.

Herniotomy is performed preferably after the age of five years at which time the delicate structures are more fully developed and the child is well stabilized. Infancy however does not interdict surgery and large herniae which are not well retained by a truss should be repaired early. Such cases are best referred to the pediatric surgeon as the frail diminutive structures require meticulous care.

**Inguinal Herniotomy**—In male children the operative procedures comprise (1) Free exposure of the sac and its highest possible ligation and extirpation (2) non transplantation of the cord and (3) musculo-aponeurotic reconstruction of the inguinal canal. The first is definitely the most important and in small herniae especially of the acquired type it is questionable whether reconstruction of the canal is at all necessary. Upon several occasions the writer has omitted uniting the conjoined muscles to Poupart's ligament with excellent results. Transplantation of the cord is neither indicated nor advisable as the cord is shortened thereby and may exert upward traction upon the testis. The Coley operation is the technic of election. Ether anesthesia is almost universally employed in special instances nerve blocking may be preferred in prepubescent children. (Refer to chapter on Anesthesia.)

**Coley Herniotomy (Bull and Coley 1892)**—Principles—Following high ligation and amputation of the sac the inguinal canal is reconstructed by suture of the conjoined muscles to Poupart's ligament in front of the cord structures the latter being replaced in their normal anatomic bed beneath the muscles. Preliminary suture of the transversalis fascia as advocated by Ferguson (1899) is rarely indicated.

**Technic**—After sterilization of the inguinal region with half-strength tincture of iodine neutralized with alcohol the skin is incised from just above the internal ring downward and inward parallel to and approximately 1 cm above Poupart's ligament. The writer prefers curving the lower portion inward to avoid the pubic area (Fig 216.) Hemostasis is secured by ligating the superficial epigastric and circumflex iliac vessel. The external oblique aponeurosis is then dissected clean of areolar tissue and the external ring defined. Care should be exercised not to divide the hypogastric nerve as it emerges through the aponeurosis just above

and to the outer side of the external ring. The wound edges are protected by saline pads held with skin clamps.

The aponeurosis of the external oblique is opened by first nicking it in the direction of its fibers opposite the internal ring and meticulously dividing the fibers down to and through the external ring. The preliminary insertion of a grooved director upward from the external ring is inadvisable as it may injure the ilioinguinal nerve. With the aid of the scalpel handle the lower flap of the external aponeurosis is swept clean of areolar tissue down to the shelving border of Poupart's ligament and the upper flap is similarly separated from the internal oblique. (Fig. 217.)

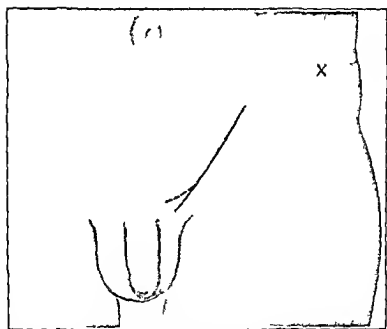


FIG. 216.—Incision for inguinal herniotomy. Curving the lower angle of the incision inward and indicated by the dotted line affords a better exposure.

*Exposure of the Spermatic Cord*—The cremasteric muscle surrounding the cord is recognized by its delicate longitudinal fibers. After these have been carefully split in order to avoid troublesome oozing, the sac and cord elements become clearly exposed without their covering of fascia propria. By grasping the latter with forceps, the spermatic cord may be lifted and stripped from the adjacent tissues for the entire length of the canal.

*Exposure of the Hernial Sac*—The hernial sac lying on the antero-mesial aspect of the vas deferens and pampiniform plexus may be identified by its pale opaque-white color. (Fig. 218.) When the sac is large, some surgeons prefer opening it and inserting

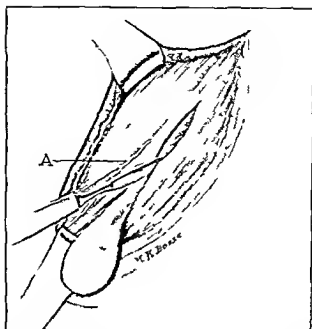


FIG. 217.—Division of the fibers of the external oblique aponeurosis exposing the inguinal canal. A Iliohypogastric nerve perforating the external oblique fiber just above and lateral to the external ring.

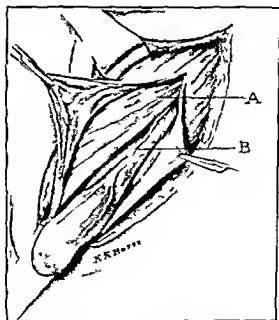


FIG. 218.—Hernial sac exposed after dividing the external oblique fibers. A Iliohypogastric nerve. B Ilioinguinal.

the index finger as a guide while separating the cord elements from it. Such procedure however is seldom necessary. The contents are readily reducible and through grasping the fundus of the hernial sac with an artery forceps the spermatic cord may be easily separated by a combination of sponging and careful dissection once the proper plane of cleavage is entered. A moistened cotton applicator is helpful in carrying the blunt dissection well up to the internal ring. Meticulous gentleness is imperative as the delicate vas deferens is easily divided and the pampiniform veins readily ruptured through careless dissection. Postoperative epididymo-orchitis is frequently in evidence of unnecessary operative trauma.

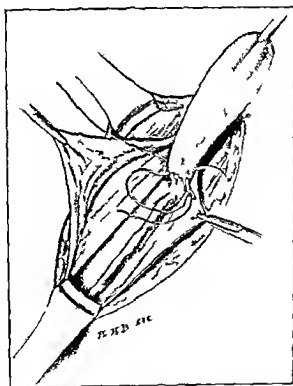


FIG 219—High transfixion ligature of the neck of the sac

*Treatment of the Hernial Sac*—While exerting slight traction upon the fundus the neck of the sac is ligated at its highest point by a transfixion ligature of No. 0 chromic catgut care being taken that the contents are fully reduced and that no muscle fibers or areolar tissue is included (Fig. 219). Upon amputation of the sac the stump will retract well within the internal ring unless the marginal muscle fibers have been embolized in the ligature.

The latter fault produces a funnel shaped protrusion of peritoneum which favors recurrence

**Reconstruction of the Inguinal Canal**—After the spermatic cord has been gently replaced in its normal anatomic bed within the cremasteric tissues the inguinal canal is reconstructed by uniting the marginal conjoint muscles (conjoined tendon) to the shelving border of Poupart's ligament in front of the cord and cremasteric muscle. Two to four interrupted sutures of No. 1 chromic catgut or silk are commonly employed. As the sutures are inserted from above downward the handle of the thumb forceps is insinuated

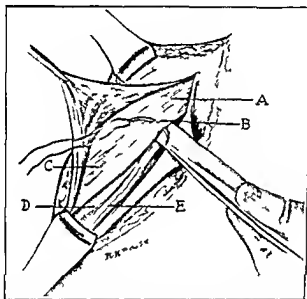


FIG. 220 Plastic repair of the inguinal canal without transplantation of the spermatic cord. The handle of the forceps is inserted beneath the conjoined muscles to avoid injury to the cord structures. A iliohypogastric nerve B margin of the conjoined muscles C internal oblique muscle D transversalis fascia E spermatic cord with ilioinguinal nerve

beneath the margin of the conjoined muscles to prevent damage to the cord structures and before the needle is passed through the shelving border of Poupart's ligament the latter is elevated from the external iliac vessels (Figs 220 and 221). The ligatures should be tied lightly to prevent ischemic atrophy of the muscle fibers. Anchorage of the lowermost suture to the pubic spine is unnecessary as the inner portion of the inguinal canal is rarely weakened in early life.

The inner and outer margins of the external oblique aponeurosis are united with a continuous suture of No. 1 chromic catgut leaving the external ring sufficient in size for the passage of the spermatic



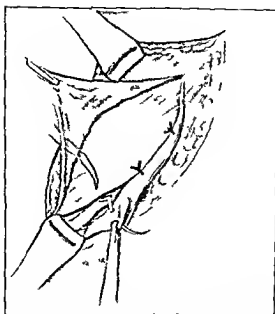


FIG. 221 — Approximation of the marginal fibers of the conjoined muscles to the shelving border of Poupart's ligament by interrupted No. 1 chromic or silk sutures.



FIG. 222 — Closure of the external oblique aponeurosis by interrupted (or continuous) sutures of No. 1 chromic catgut.

cord (Fig 222) In cases of large herniæ, the excessive portion of the external oblique aponeurosis may be imbricated The skin edges are approximated with dermal, silk or plain gut sutures The wound is then powdered with thymol iodide and the gauze dressings covered with oil silk or rubber tissue to avoid soiling in voiding A scrotal bridge is unnecessary and adds to the child's discomfort

**Bilateral Herniotomy**—This is indicated whenever weakness is evidenced in the opposite inguinal canal In most instances a patent funicular process of 1 cm or more is demonstrable High ligation and amputation of the incipient bubonocoele is sufficient to prevent the subsequent development of hernia without reconstruction of the canal This prophylactic procedure requires but a few minutes and in no way influences the morbidity or operative risk (Some surgeons routinely elect bilateral herniotomy when a large congenital hernia is present)

**Herniotomy in Females**—After the inguinal canal has been exposed the hernial sac should be meticulously dissected from the round ligament in order to preserve the latter in its anatomic bed for uterine suspension When divided through accident, the stump of the ligament should be sutured to Poupart's ligament at the internal ring without traction as the latter may produce uterine displacement The inguinal canal is then closed throughout its entire length by suturing the conjoined muscles to the shelving border of Poupart's ligament and approximating the edges of the external oblique aponeurosis

**Operation for Direct Hernia**—The procedure is the same as that for indirect hernia except the cord is transplanted and the inguinal canal is reconstructed with greater stability While the cord structures are gently retracted and held aside by a strip of moistened tape the bulging herniation through the transversalis aponeurosis is repaired by reefing the latter with No 1 chronic catgut without opening the sac The margins of the conjoined muscles are then accurately approximated to the shelving border of Poupart's ligament for its entire length the lowermost suture passing through the pubic periosteum for firm anchorage Should this procedure produce too great tension upon the conjoined muscles, transplantation of the rectus muscle may be employed (Halsted) The lateral margin of the rectus sheath is opened and the muscle is retracted outward and sutured to the inner portion of the shelving border of Poupart's ligament

After completion of the musculo-aponeurotic reconstruction of the floor of the canal the cord may be replaced upon the internal oblique muscle and the external oblique aponeurosis united over it, or the margins of the aponeurosis may first be accurately united down to the pubic spine and the cord then allowed to rest upon it In the latter procedure the external ring will be almost opposite the

internal. In placing the spermatic structures in their new bed torsion of the cord should be carefully avoided.

**Postoperative Treatment Following Herniotomy**—The child should be well blanketed when returned to bed and chilling from exposure carefully avoided. Water and fruit juices may be given as soon as the postoperative nausea has subsided. In the interim 10 per cent glucose solution may be administered by proctoclysis. In cases of excessive nausea and vomiting the saline water balance should be maintained by phlebotomies or hypodermoclyses of 3 per cent glucose in physiologic saline solution.

Codem and the barbiturates are administered respectively for postoperative pain and restlessness. Catheterization for retention is seldom necessary. Full diet is resumed by the fourth day and the bowels are moved by enemata *per rectum*. The patients are permitted to move freely in bed and it is not unusual for young children to sit in their cribs on the second or third day. This is inconsequential and less harmful than restraint with attendant crying and struggling. Young children are allowed to walk on the eighth day and older ones on the tenth. Vigorous exercise should be interdicted for six weeks.

**Complications Following Herniotomy**—Postoperative complications are much less frequent in children than in adults. *Wound infections* are generally superficial and occur in approximately 2 per cent of the cases chiefly from wetting with urine. If heavy chromic kangaroo tendon or non absorbable suture material is employed the knots may occasionally produce a serous discharge until extruded. *Epididymo-orchitis* results from operative trauma and is largely preventable. In rare instances it is followed by testicular atrophy. *Postoperative pneumonia* may be minimized by several measures: nasopharyngeal prophylaxis, avoidance of operation when upper respiratory infections are prevalent, the gentle handling of tissues and skilfully administered anesthesia. The incidence of pulmonary complications appears to be reduced by autohemotransfusion. Following operation 2 to 5 cc of blood are withdrawn from the median basilic vein and immediately reinjected into the tissues of the buttocks.

**Results of Operation**—Inguinal herniotomy is one of the safest and most satisfactory of surgical procedures and permanent cure obtains in practically all uncomplicated cases.

### FEMORAL HERNIA

Rupture through the crural canal rarely occurs in early life and only about 1 per cent of all femoral herniæ develop during childhood. The condition is more common in the female in the approximate ratio of 3 to 1 and is twice as frequent on the right side.

The hernia is bilateral in about one-fifth of the cases and at times may be associated with the inguinal variety.

**Anatomy**—The herniation occurs through the Femoral or Crural Ring. This is bounded in front by Poupart's ligament and the deep crural arch behind by the ileopectineal line of the os pubis, the bone being covered by the pectineus muscle, pubic portion of the fascia lata and by Cooper's ligament, mesially by Gimbernat's ligament and laterally by the femoral vein. The crural ring constituting a weak point in the abdominal wall is closed by a layer of compact areolar tissue, the Septum Crurale. The upper surface of the latter is concave and a shallow depression presents on the inner surface of the peritoneum termed the femoral fossa. As the hernia evaginates the septum crurale is pushed ahead of it and forms one of its coverings.

The Femoral Canal through which the hernia descends is a narrow space within the femoral sheath just mesial to the femoral vein. In childhood it is less than 1 cm. in length and extends from Gimbernat's ligament to the upper part of the saphenous opening. Upon reaching the latter the hernial sac traverses anteriorly by pushing the cribriform fascia before it and presents beneath the skin just below Poupart's ligament. The coverings of the hernia consist of the following structures: peritoneum, properitoneal fat, septum crurale, femoral sheath, cribriform fascia, superficial fascia and integument. The structural confines of the femoral ring are elastic in early life and strangulation rarely develops.

**Etiology**—Although controversial the saccular theory has many advocates. It is contended that in certain instances the parietal pelvic peritoneum adjacent to the femoral vessels adheres to them and is drawn through the femoral canal during the longitudinal growth of the thigh. Predisposing factors favored by others comprise congenital defects of Gimbernat's ligament or of the septum crurale, broadening of the female pelvis and long-continued abdominal strain.

**Symptomatology**—The hernial sac is small and globular and the neck narrow. The usual content is omentum which at times becomes adherent to the sac through inflammation. Occasionally small intestine also evaginates into the sac either as a loop or partial enterocoele (Richter's hernia, Fig. 223).

**Diagnosis**—Femoral hernia evaginates below Poupart's ligament and lateral to the pubic spine. In the presence of reducibility the differential diagnosis from inguinal hernia may be made in the following manner: if the external inguinal ring and canal are protected by finger pressure, a femoral hernia will recur upon straining or coughing. In obese female children the diagnosis may be difficult. *Enlarged femoral lymph nodes* are accompanied by symptoms of inflammation and exhibit a primary focus of infection. *Proximal vessels*

may be distinguished by the presence of a fluctuating mass upon deep manual pressure in the inguinal region and by a spinal roentgenogram. Lipomas are superficial lobulated tumors often connected with the skin and fail to exhibit expansile cough impulse.

**Treatment**—In the presence of reducibility a supportive truss should be given a trial although the results are seldom curative. Operation is indicated in cases of irreducibility, inflammation and strangulation and in older children in whom a truss has proven ineffective.

**Femoral Herniotomy**—This comprises (1) removal of the sac and (2) closure of the femoral ring and femoral canal. Bassini (1894) advocated the latter by suture of Poupart's ligament to the pectineal fascia (Cooper's ligament) and of the falciform process to the pubic portion of the fascia lata. This technique has been most widely subscribed to with highly satisfactory results. The inguinal approach however first described by Annandale (1876) and later modified by Gordon (1900) and Moschowitz (1905) has definite advantages in children and is preferred by the author. Ochsner and others contend that high ablation of the sac will result in cure and that closure of the femoral ring and canal is unnecessary.

**A Femoral Operation**—The skin incision 3 to 5 cm. in length is made just below and parallel to Poupart's ligament. (Some prefer a vertical incision equidistant above and below the hernial swelling.) The fascia lata is freed of all fatty tissue thereby exposing Poupart's ligament and the falciform process of the fascia lata. The saphenous vein should be carefully avoided. The sac often covered with peritoneal fat is carefully dissected from the surrounding structures care being taken not to injure the femoral vein which lies close to the outer wall. The neck of the sac is freed well up to the femoral ring by blunt dissection. If empty a transfixion ligature is applied as high as possible while exerting slight traction upon the sac. Should the latter contain irreducible contents it is opened at the fundus and the adherent omentum or intestine is carefully separated and replaced within the abdominal cavity. The sac wall

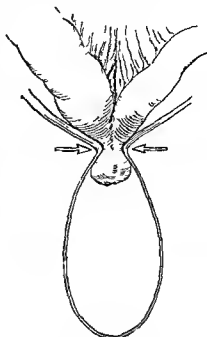


FIG. 223. Right-sided hernia or partial enterocoele.

is very thin and any unusual thickness should suggest the possible presence of the bladder or of a sliding hernia

If the sac has been well freed of all adventitious coverings the ligated stump will disappear from view after the sac is amputated. The femoral ring may be closed as follows: A No. 1 chromic purse-string suture is passed through Poupart's ligament from before backward just mesial to the site of the femoral vein; the needle is then inserted through Cooper's ligament (pectineal fascia) from without inward and finally is insinuated through Poupart's ligament so as to emerge near its original entrance (Fig. 224). Upon tying the suture the femoral ring and upper portion of the canal become closed. A single purse-string suffices. The integument is then approximated with silk, plain catgut or dermal suture.

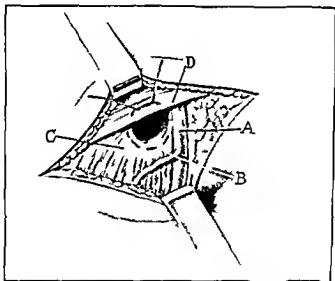


FIG. 224.—Closure of the femoral ring by a purse-string suture which approximates Poupart's ligament to Cooper's ligament and the pectineus muscle and fascia. A Femoral vein B Cooper's ligament C pectineus fascia D Poupart's ligament

**B Inguinal Operation**—The inguinal canal is exposed by dividing the external oblique aponeurosis in the same manner as in inguinal herniotomy. While the upper and lower borders of the external oblique are held aside the cord structures and conjoint muscles are retracted upward, thereby exposing Hasselbach's triangle. The transversalis fascia forming its floor is then split from the pubis to the region of the deep epigastric vessels (Fig. 225). Upon reflecting the edges the neck of the hernial sac is seen at the femoral ring. Any adherent omentum or intestine is carefully dissected from the

wall of the sac and the latter is withdrawn into the abdomen. The neck is then ligated with a transfixion ligature and the sac amputated.

In cases of strangulation of the intestine, the latter may be reduced by enlarging the neck of the sac through division of Gimbernat's ligament from without inward.

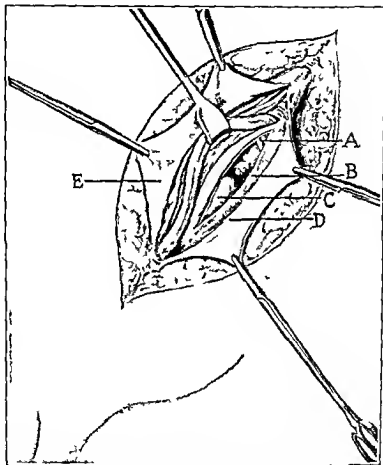


FIG. 225.—Exposure of a femoral hernia through the inguinal canal. *A*, Deep epigastric vessels; *B*, incised transversalis fascia; *C*, sac of femoral hernia; *D*, Poupart's ligament; *E*, aponeurosis of external oblique.

The femoral ring is closed by a No. 1 chromic purse-string suture passed in the same manner as in the femoral operation. The inguinal canal is then reconstructed without transplantation of the cord (Coley technic of Herniotomy).

The end results of both types of operative repair are equally good and practically all cases are permanently cured. Complications are unusual. (Refer to Inguinal Herniotomy.)

## SLIDING HERNIA.

In sliding hernia, the extraperitoneal portion of the cecum or ascending colon, or of the sigmoid or descending colon, protrudes through the internal abdominal ring and forms a part of, or at times, the entire hernial protrusion.

The etiology and production of sliding herniæ remain obscure. The condition occurs in approximately 1 per cent of all adult inguinal herniæ but is rare in childhood.

**Clinical Findings.**—The sliding gut may replace a portion of the sac or the hernia may be sacless. (Fig. 226) In the latter instance,

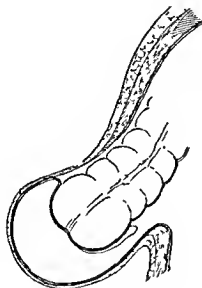


FIG. 226 —Sliding hernia. Note peritoneal reflection

the intestinal wall may be mistaken for a thickened hernial sac and be inadvertently opened. In case of doubt, the incision should be extended so that the peritoneal cavity may be opened at a safe distance from the hernia. The same precautionary measures should be instituted when the intestine is incorporated in one side of the sac for attempts to separate it may damage the circulation of the bowel. A sliding hernia should always be suspected when the intestine separates with difficulty; in irreducible enterocele such separation is readily performed without bleeding.

**Treatment.**—Since a sliding hernia is never completely reducible, truss support is definitely contraindicated. After exposing

the inguinal canal, the treatment of the sac and its contents requires special consideration. In small sacless types, the protruding gut may be reduced completely within the abdominal cavity. In the reparative herniotomy of such cases by the Coley technic, the weakened and enlarged internal ring should be firmly reconstructed. In instances where the spermatic vessels and vas deferens are widely separated by the herniating bowel, two internal rings may be established by passing a separate stitch through the conjoined muscles and Poupart's ligament between the structures.

When the bowel wall is intimately associated with the sac, the safest procedure is to enlarge the incision outward and open the peritoneal cavity at a safe distance from the hernia. The evaginated intestine is then retracted into the peritoneal cavity and a sufficient



area of peritoneum in the iliac fossa is denuded for anchorage of the cecum or colon (cecopexy or colopexy). Hotchkiss recommends opening the sac and utilizing the flaps for peritonealization of the herniated bowel but such procedure is seldom applicable in children. Recurrences following any type of plastic repair are not uncommon.

### EPIGASTRIC HERNIA

In epigastric hernia the evagination occurs in the linea alba above the umbilicus. The condition rarely develops in infancy and is uncommon before the age of twenty years. Males are more commonly affected.

**Anatomy**—The linea alba is formed by the mid line fusion of the aponeuroses of the three flat abdominal muscles. In the lower abdomen the recti muscles are closely approximated and the linea alba is narrow and firm. In the upper portion however the recti are farther apart and the linea alba is wider and thinner. Potential mid line weakness is further favored by the prolongation of the transversalis fascia along each of the several blood vessels which penetrate the linea alba. The intimate association of the fatty elements of the falciform ligament to the linea alba may also be a predisposing factor.

**Pathology** Epigastric herniæ are always small and the ring is rarely 1 cm. in diameter. The evagination consists of protruding adipose tissue (lipoma) of the falciform ligament and a sac is seldom present. Occasionally a funnel like process of peritoneum is dragged outward by the herniating fat and this may contain omentum. Irreducibility of the contents is favored by the small opening in the linea alba but strangulation rarely develops.

**Symptomatology** The majority of epigastric herniæ are asymptomatic. Focal pain and tenderness may develop infrequently from either trauma or pinching of the lipoma by the narrow ring. Although many abdominal complaints have been attributed to epigastric hernia reflex abdominal symptoms are seldom produced thereby.

**Treatment**—None is required in asymptomatic cases. An abdominal support may actually aggravate the condition. Operation should only be recommended for persistent pain and tenderness.

**Operative Technique**—A small mid line incision through the skin readily exposes the shining covering of the fatty nodule. After the fat has been teased apart to exclude the possibility of peritoneal protrusion the nutrient vessels are ligated and the lipoma amputated. The stump is then replaced through the aponeurotic opening and the hiatus closed by overlapping the edges with No. 2 chromic catgut sutures.

## INCISIONAL HERNIA (POSTOPERATIVE HERNIA)

The dominant factor in the production of incisional hernia is wound sepsis. Although improved suture material and the less frequent use of intraperitoneal drainage in the presence of diffuse peritonitis have lessened the tendency to mural infection the present day incidence of postoperative hernia in cases of perforative appendicitis approximates 10 per cent.

Sloughing of the rectus sheath or external oblique aponeurosis is the most common cause of postoperative hernia. This occurs more often in the gridiron type of incision than in the split rectus. Hernia may also develop when the fascia remains viable through the evagination of omentum about the drain or into the hiatus when the drain is removed.

Hernia also occurs in a small percentage of clean cases. Improper peritoneal closure which permits a tab of omentum to evaginate into the wound is the most frequent cause, also the untying or breaking of sutures through excessive distention or vomiting may permit a tab of omentum or a loop of gut to herniate. The latter may cause mechanical ileus and if unrecognized prove fatal. Many such deaths are falsely attributed to postoperative paralytic ileus. Wound inspection should always be made in cases of persistent vomiting or distention of doubtful origin and particularly when a serosanguinous discharge appears on the dressings. Upon separating the wound margins a loop of gut may be found within the deep confines of the wound.

The failure to wear an abdominal support in the presence of a weakened wall may lead to the gradual development of a hernia. Injury to the lower dorsal nerves resulting in muscle atrophy produces a diffuse bulging rather than actual herniation.

**Pathology** — The absence of a true hernial sac in incisional hernia supports the hypothesis that herniation usually occurs immediately or soon after operation from the evagination of omentum or gut through an opening in the line of peritoneal closure. The dome of the hernia often presents a false sac of condensed areolar tissue which is adherent to the surrounding mural structures. The underlying firmly attached omentum may include intestine.

**Treatment** — Operation should be advised early before the incipient hernia becomes large and more difficult to repair. The skin incision requires caution as the omentum or gut may be subcutaneous. The contents are then carefully separated from the surrounding structures and replaced within the abdominal cavity. A perfect anatomic repair may be obtained through free dissection of the mural structures and separate tier suture of the peritoneum and posterior rectus sheath, rectus muscle and anterior sheath.

## DIAPHRAGMATIC HERNIA

Since the embryology and anatomy of the diaphragm are fully described in text books on these subjects mention will be made only of certain factors which concern the development of diaphragmatic hernia.

**Normal Apertures**—The normal apertures of the diaphragm through which structures pass between the thoracic and abdominal cavities consist of the following (1) Aortic for the passage of the aorta thoracic duct and azigos major veins, (2) caval for the inferior vena cava and (3) esophageal for the esophagus and vagi nerves. The last is the only normal opening through which diaphragmatic hernia may develop.

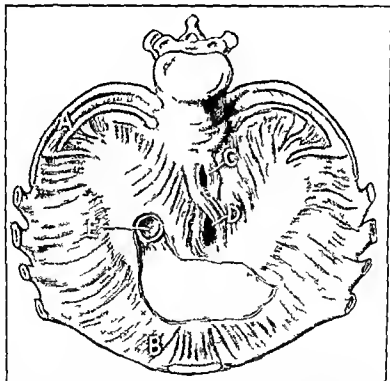


FIG 227 View of the diaphragm from above. A Foramen of Bochdalek. B foramen of Morgagni. C, D and E aortic esophageal and caval openings.

**Accessory Apertures** Other openings or weak zones may result from fusional failure of the pleuroperitoneal membranes at the following sites (1) The foramen of Morgagni. This common point of weakness presents as a triangular cleft (Larrey's space) between the sternal and costal attachments of the diaphragm (Fig 227). (2) The foramen of Bochdalek. This is a similar hiatus

in the posterior region between the vertebral and costal diaphragmatic regions. The membranes close last at this site and it is a frequent point of weakness. (3) Large areas of muscular deficiency in the central portion of the diaphragm occur occasionally from developmental failure of the musculature between the central and costal regions.

**Weak Zones** The central areas of the right and left domes of the diaphragm constitute its weakest portion. The right dome is well buffered by the liver, the left however covers only a small portion of that organ and lies above the stomach and coils of intestine. The latter while undergoing rotation are crowded in this area. It is through this central portion of the left dome that congenital herniae develop, the ectopia of the viscera occurring at the time of diaphragmatic formation. For similar reasons traumatic hernia of the diaphragm due to excessive pressure over the abdomen is eight times more frequent in the left unprotected zone.

**Nerves of the Diaphragm** The phrenic nerve supplies the muscle buds of the embryonic diaphragm and follows the descent of the primitive organ from its position under the fourth and fifth cervical segments. Sympathetic fibers from the lower cervical ganglia accompany the phrenic. Pursuing an antero-lateral course and piercing the diaphragm between the muscular and tendinous portion each phrenic nerve under cover of the peritoneum divides into three branches, anterior, lateral and posterior. Filaments from the seven lower intercostal nerves to the rim of the diaphragm have also been described. Although a dual phrenic and costal innervation is thus indicated, division of the phrenic fibers results in atrophy of the diaphragmatic musculature.

**Varieties of Diaphragmatic Hernia** — Diaphragmatic hernia may be congenital, traumatic or acquired. The esophageal opening is the most common site of the congenital type. A true hernial sac is often present and the stomach is the organ commonly herniated. Acquired herniae appear in those parts of the diaphragm which are subject to developmental weakness and thus suggest a congenital origin. For this reason some investigators classify all diaphragmatic herniae as either congenital or traumatic.

**Contents of the Hernia** — Almost every abdominal organ has participated in diaphragmatic herniation. Hedhlof reports one case in which even the kidney was found in the pleural cavity. When the contents consist of stomach, intestines and omentum the usual arrangement is as follows: the stomach occupies a posterior position, the colon a mid position and the coils of the small intestine an anterior one. (Fig. 228.)

**Symptomatology** Diaphragmatic hernia may exist a lifetime without symptoms. The latter depend upon the nature of the pathology and may vary from a mild cough or slight degree of

indigestion to severe dysfunction of the heart, lungs and digestive tract. If the stomach alone is involved and the cardia slides up and down the condition may escape detection until adult life. When the entire stomach passes into the pleural cavity its distention with fluid and gas may produce dyspnea, cyanosis, rapid pulse and respiration, cough and vomiting. Dysphagia may also occur from torsion of the lower end of the esophagus. Extreme eventration may result in still birth.

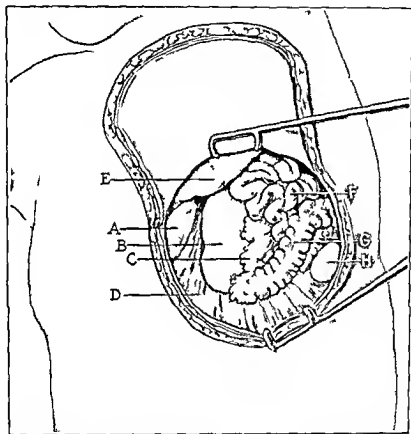


FIG 273 —Contents of the pleural cavity exposed through a laparotomy. A Heart B stomach C omentum D diaphragm E lung F small intestine G colon H spleen

Cyanosis occurring soon after birth is a frequent symptom. It differs from that of cardiac disease in that the attacks are spasmodic in character and are often produced by feeding or crying. Prompt relief may follow emesis or the changing of the child's position from the recumbent to the erect. Cough when present, is also paroxysmal in type and the seizures are almost always relieved by placing the patient upright.

**Diagnosis** The physical signs of diaphragmatic hernia are elusive and bizarre varying from a total absence to those of pneumothorax hydrothorax or pneumonia. Litten's sign is absent. Dextrocardia may be present in some degree when the hernia is on the left side and is of considerable magnitude. A positive diagnosis is readily made through the aid of serial roentgenograms after an opaque meal. A roentgen ray of the colon after a barium enema should also be taken to define the location of the bowel. Fluoroscopic examination is a further diagnostic aid.

**Treatment**—Palliative treatment should be directed toward the prevention of gas in the stomach and colon through careful feeding and the administration of antifermentatives. A change to the erect posture will often relieve a paroxysm of coughing or cyanosis. Operation only becomes indicated when the symptoms are recurrent and progressive.

**Diaphragmatic Herniotomy** Preoperative preparation of the patient is highly important. Gaseous distention should be reduced to a minimum through diet and enemata. When the colon is involved and distention persists a preliminary appendicostomy is advisable. An extra intake of carbohydrates should be administered for a few days prior to operation in order that glycogen storage in the liver and muscles may be at its maximum. When impractical by oral administration extra sugar may be introduced in the form of glucose in saline solution by phlebotomies or hypodermoclyses.

**Anesthesia** Since adequate relaxation is necessary to insure retention of the hernial contents within the abdominal cavity during the plastic repair of the diaphragmatic hiatus a skilfully administered anesthetic is of prime importance. The safest and most satisfactory agent is either gas-oxygen-ether or gas-oxygen-cyclopropane under carefully measured pressure. Through pressure anesthesia the lung may be inflated or deflated without danger of asphyxia. A soft pillow placed under the healthy side aids the respiratory movements. Some operators prefer to support the patient in a sitting posture by means of a right-angled seat attached to the operating table.

**Methods of Approach**—Although the hernia may be approached through either a transthoracic or transabdominal incision or a combination of both the transthoracic has definite advantages. Adherent hernial contents may be more safely reduced and the hiatus repaired more satisfactorily.

**Operative Technique** A lipel incision is deepened down to the ribs and the pleural cavity opened. (See Fig. 228.) While a shield director is inserted to protect the hernial contents the ribs are divided at each extremity by a costatome and the trap-door flap is turned upward. The hernial contents are then gently held aside while the phrenic nerve is identified and anesthetized with 2 per cent novocaine to secure diaphragmatic immobilization. The result

ing transient paralysis provides not only additional working space but aids greatly in the reduction of the hernia and in suturing the hiatus.

After the contents have been restored within the abdominal cavity the opening in the diaphragm is closed by means of a purse-string silk suture reinforced with two or more mattress sutures (Silk holds well in the presence of pleural effusion). The osteoplastic flap is then turned down and sutured with silk worm so as to accurately approximate the pleural edges and the rib ends. Before the final suture is applied the lung is inflated with CO gas until it reaches the diaphragm.

**Postoperative Treatment**—Pressure inhalations of CO are administered every four hours for five minutes during the first three postoperative days in order to minimize the danger of pleural effusion. To prevent upward pressure upon the diaphragm a stomach tube should be passed at the first indication of gastric distention.

## INTERNAL HERNIA

### (RETROPERITONEAL HERNIA)

In the changes which the abdominal contents undergo from early embryonic to adult arrangement certain peritoneal folds fossæ and the foramen of Winslow are normally produced. Additional abnormal openings or fossæ may be formed through faulty development. Coils of small intestine may herniate into any of these and give rise to internal hernia.

The regions in which such evaginations may occur are (1) About the duodeno-jejunal junction (2) the cecum (3) the mesentery of the sigmoid and (4) the foramen of Winslow.

**Duodeno jejunal Fossæ**—In the neighborhood of the ligament of Treitz where the small intestine passes from a retroperitoneal to a mesentery supportive structure the peritoneum may be thrown into folds resulting in fossa formation. Moynihan has described nine such fossæ the superior and inferior horns of the duodenal fossæ on the left side of the ascending duodenum being the most constant. The paraduodenal fossa of Landzert lying to the left and a little distance from the duodenum results from the raising of a peritoneal fold by the inferior mesenteric vessels. The mesocolic fossa is formed by a peritoneal fold containing the ascending branch of the left colic artery. The mesenterico-parietal fossa lies near the first part of the jejunum behind the superior mesenteric artery.

**Paracecal and Other Fossæ**—Several fossæ are also described about the cecum the ileocolic lying above the ileocecal valve the ileocecal behind the valve and the retrocecal behind the cecum and meso-appendix. The intersigmoid fossa lies between the root of the mesentery of the sigmoid and the parietal peritoneum. All of the foregoing occur inconspicuously and vary in size from a shallow dimple to that of an actual peritoneal fossa. The foramen of

Winslow is quite small in early life and scarcely admits the tip of the little finger

**Symptomatology**—The symptoms of internal hernia are either absent or so vague that the diagnosis is seldom made until acute intestinal obstruction develops. In cases of the latter without apparent cause the possibility of internal hernia should always be considered.

**Treatment**—The pathology is generally discovered during exploratory laparotomy for acute intestinal obstruction and the surgical therapy comprises (1) Reduction of the hernia (2) appropriate treatment of the damaged gut and (3) closure of the fossa when possible. Reduction of the contents may be a difficult problem when the constricting ring includes vital structures. This is especially true of hernia through the foramen of Winslow or into the fossa beneath the mesenteric vessels. If distention of the gut is such as to prevent reduction the safest procedure is to open the peritoneal sac beyond the neck. Reduction may then be attempted through pressure manipulation of the distended loops. Failing in this the obstructed bowel may be evacuated of its contents through a temporary ileotomy which is immediately closed. The collapsed intestine is then reduced through the hernial opening and its viability carefully determined. When gangrenous resection with side-to-side anastomosis is best performed. If closure of the neck of the fossa is impractical its obliteration may be accomplished through marsupialization with gauze.

### RARE FORMS OF HERNIÆ

#### (LUMBAR OBTURATOR SCIATIC PERINEAL)

Each type will be but briefly described. Their occurrence is exceedingly rare and the writer's experience is limited to a case of hernia through Petit's triangle in a male child of seven years.

**Lumbar Hernia**—Spontaneous lumbar hernia appears as a protrusion in the lateral abdominal wall between the costal margin and the iliac crest. Herniation may occur through the following areas: (1) The inferior triangle described by Petit (1738) bounded in front by the external oblique muscle behind by the latissimus dorsi and below by the iliac crest (2) the superior triangle of Grunfelt Leashaft (1866) or (3) at any other point of weakness in the lumbar region.

The condition may be congenital or acquired and in the latter instance develop spontaneously or subsequent to trauma. Any developmental defect of the muscles upon the ribs may be responsible for the herniation. Watson was able to find only 116 cases in the literature and the ages of greatest incidence were between two and ten years and after fifty years. The majority occurred through either the inferior triangle of Petit or the superior triangle of Grunfelt Leashaft. A hernial sac was



generally present which contained omentum or fat, and infrequently the appendix, stomach, cecum, sigmoid or kidney. Eighteen cases were congenital.

Acquired lumbar hernia may result from some developmental defect in the presence of undue strain or from trophic muscular changes following poliomyelitis, Pott's disease or trauma.

**Treatment**—Herniotomy is usually indicated since support by a truss or other mechanical means is rarely satisfactory. In exposing the hernia, caution should be exercised in separating the fatty mass in order to avoid possible injury to the colon or other hollow viscus. In hernia through Petit's triangle, the method of repair described by Dowd is very satisfactory. After reduction of the hernia and ablation of the sac, a flap of fascia lata taken from below the ileum is turned upward and sutured to the latissimus dorsi muscle behind, the external oblique in front, and the lumbar fascia above. A flap of sheath and fascia from the latissimus dorsi is then reflected forward over the first transplant and sutured to the external oblique. In herniae occurring at a higher level some form of musculo-aponeurotic repair is usually adaptable.

**Obturator Hernia**—In this exceedingly rare type of hernia, protrusion occurs through the weak portion of the obturator foramen which provides passage for the obturator vessels and nerve. Three varieties have been described in which the process of peritoneum protrudes along these structures. In the commonest type, the sac emerges through the obturator canal and follows the anterior branch of the vessels to lie in front of the obturator externus muscle under the pectineus. The second variety follows the inferior vessels and presents between the middle and superior fasciculi of the obturator muscle. In the third form the sac lies between the obturator membranes behind the obturator muscle.

**Treatment**—Preoperative diagnosis is seldom possible and the condition is generally discovered during exploratory laparotomy for acute intestinal obstruction. After reduction of the contents and appropriate treatment thereof, the sac is withdrawn into the pelvis, its neck ligated and the sac amputated.

**Sciatic Hernia**—Sciatic hernia (*gluteal, ischiatic*) is the rarest of all forms. Watson was able to collect only 30 cases and in 6 the condition was congenital. Three varieties have been described. In 2 of these, herniation occurs through the greater sacrosclatic foramen. Depending upon whether the evagination is above or below the pyriform muscle, they are termed respectively suprapyriformis and subpyriformis. The third and least common type passes through the lesser sacrosclatic foramen.

The hernia may present as a small swelling in the gluteal region or its presence may remain unsuspected until strangulation necessitates exploratory laparotomy. In the former instance a roentgenogram may be helpful in defining the hernial shadow.

## PART VIII

# UROLOGIC CONDITIONS

By CLARENCE G. BANDLER, M.D., F.A.C.S. AND  
ALBERT H. MILBERT, M.D.

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### CHAPTER XXXIII

#### GENERAL CONSIDERATIONS

THE past decade has witnessed evolution of pediatric urology as a specialty within a specialty. In this relatively short span of time the diagnostic refinements and therapeutic advances made in the study of genito-urinary diseases during the past twenty-five years have been applied in turn to solving the urologic problems of childhood.

In so doing the barrier of ignorance and sentimentality has been torn down and in its place a rational approach to solving the all too frequently overlooked juvenile genito-urinary tract lesion has been substituted. The amassing of a wealth of pathologic and clinical findings together with therapeutic successes abetted by the demonstration of the value and innocuousness of an urologic investigation has convinced the most skeptic practitioner and pediatrician. It has led to a dissipation of his previous reluctance to submit tiny patients to instrumentation and to a sharpening of his diagnostic acumen in the recognition of an obvious or latent lesion.

Similarly the urologist now approaches the problem of treating the child without the timidity and sense of limitation he had previously experienced. The introduction of intravenous urography was a boon to initiating urologic study in a greater number of juvenile patients. The perfection of instruments representing miniatures of the tried and proven adult armamentarium forged the final link and what has been wrought will be presented in the ensuing pages.

**The Diagnostic Approach**—It has been stated facetiously yet truthfully that the greatest of medicos are the pediatrician and the veterinarian. In most cases they must diagnose and treat without the assistance of their patients. As a result of the inarticulateness of the young one is dependent upon the observations of the parent

masked by nausea vomiting or constipation. This reflex gastrorenal and intestinal renal interrelationship must be constantly kept in mind.

An inventory of the various systems is essential since remote complaints referred to the chest limbs or skull may be the initial manifestations of distant metastases of highly malignant genitourinary tumors. Previous operative intervention should be noted. More than one appendectomy has been performed when the trouble really was confined to the urinary tract as a pyelonephritis or infective process engrafted on a congenital abnormality. We say this with no malice toward the general surgeon since the differential diagnosis is frequently difficult and it is far wiser to explore than to watch a peritonitis develop as a result of overcaution.

**Symptomatology**—Gross external deformities are readily discernible by the obstetrician midwife nurse or parent. *Masses* in the hypogastrium or loin make their presence known to the guardian by altering the body's symmetry. *Frequency of micturition and nocturia* except as they reflect a response to undue ingestion of fluids as in diabetes mellitus are expressive of urinary tract irritation. Only by complete investigation can the cause be determined as functional or organic due to infection congenital abnormality lithiasis or a neurogenic lesion.

*Straining or painful urination* may point to lower tract obstruction—neurogenic vesical pathology congenital posterior urethral valves hypertrophy of the verumontanum or urethral stricture. *Incontinence* or so-called *enuresis* manifesting itself as bed wetting at night or clothes wetting diurnally is a frequent source of complaint when it persists after the age of four years. While over 90 per cent of these cases are functional in origin the persistence of symptoms in the face of assiduous medical care warrants urologic study. As a result frank surgical lesions such as irritating vesical or urethral polypi or excrescences and even renal tuberculosis have been disclosed.

*Hematuria* is always an alarming discovery to the parent. The child may think he has done something wrong and fail to report it. The nurse may find the diaper of the infant saturated with a bloody urine. More often the hematuria is microscopic and comes to the attention of the physician through the medium of a routine urinalysis. *Pyuria* unless grossly manifest is likewise discovered in the laboratory.

*Pain* may be linked with crying during micturition or be reported by the older child. The absence of urinary findings should not terminate the urologic examination. Dull pain in the loin may be the only tangible evidence of a growing neoplasm pyogenic abscess tuberculous focus or hydronephrosis.

*Fever* is commonly associated with urogenital infections, either *acute or chronic in nature*. A long continuous or remitting febrile course, otherwise unexplained, may find its source in the urinary tract. Chills or convulsions may occur, depending on the degree of uresepsis.

Recurrent *gastro-intestinal upsets, malaise, weakness, pallor, loss of weight, or undue drowsiness* may, either individually or collectively, point to a chronic suppurative focus, a slow-growing neoplasm or an advancing renal lesion with impending insufficiency and uremia.

**Physical Examination.**—Because of the frequent inadequacy of the history, objective findings assume a greater importance and their recognition is essential to diagnosis. While a preliminary general physical examination usually precedes the urologic investigation, the urologist checks these systemic findings as they apply to the local examination.

The patient's state of nutrition, color and mental status are worthy of note. The characteristic build of the Froelich type is associated with genital hypoplasia and late or maldescent of the testes. Hirsutism or abnormal genital or mammary development may be the first outward indication of a tumor of the pituitary or adrenal gland. Cutaneous pyogenic lesions not infrequently antedate and are directly responsible for suppuration in the urinary tract.

**Examination of the Abdomen**—This should be approached in a systematic manner. Inspection may reveal an asymmetry in the flank, mid-abdomen, or hypogastrium. A large hydronephrosis, renal tumor, or chronically distended bladder may be the underlying cause. The umbilicus may be the site of a deep-seated inflammation or be the source of a numerous secretion associated with a patent urachus. Distrophy of the bladder is self-evident. Palpation and percussion will prove further aids in diagnosing any abnormality noted on inspection. Needless to say, judgment on a hypogastric tumor should be deferred until after the bladder has been emptied by catheterization. Masses not discernible by inspection may be encountered on palpation. Bimanual kidney palpation should always be done with the patient supine and the knees and thighs flexed on the trunk. Relaxation may be further effected by examining the child while immersed in a warm bath. In the interpretation of abdominal masses one must bear in mind the incidence of abnormal development and position of the kidney. A congenital solitary kidney or a fused one may be palpated in the mid-line, while a ptosed or ectopic organ may be felt in the pelvis. Cystoscopy and urography confirm or refute these findings.

The male external genitalia should be studied carefully. *Inspection* reveals variations in development or any deviation from the

normal of the penis or scrotum. Anomalous injuries, superficial inflammations or tumors are noted in this way. Phimosis or paraphimosis, hypospadias or epispadias, scrotal cysts, hernia or inflammations may be evident at a glance. Palpation of the scrotal sac directed at determining the condition of the testicular tumors, the testicle proper, the epididymis and the spermatic cord is next undertaken. All scrotal masses should be transilluminated to rule out encysted clear fluid tumors. Any urethral discharge is abnormal and warrants immediate bacteriologic investigation.

The female external genitalia should likewise be subjected to close scrutiny. Inspection of the labia clitoridis and external meatus may disclose anomalies, inflammatory adhesions or tumors. The finding of an ectopic ureteral orifice close to the vaginal orifice has explained several cases of puzzling urinary incontinence. In the young, rectal palpation should be substituted for bimanual vaginal examination.

The inguinal and femoral regions should not be overlooked and any inguinal or femoral adenopathy, tubercle formation or herniation noted.

Rectal examination together with bimanual abdominal palpation completes the routine examination of the child from an urologic standpoint. Because of common embryologic origin, anomalies of the external genitalia may be accompanied by anal or rectal defects. Tumors of the internal genitalia in either sex may be first recognized by the examining finger in the rectum.

**Urinalysis** — The examination of the urine assumes great importance since it represents the one accurate means of determining the functional status of the urinary tract. While a negative urinalysis does not rule out genito-urinary tract pathology, the urine reflects the presence of an abnormality when present in the majority of cases. Chemical, microscopic and bacteriologic study of repeated urine specimens should precede the cystoscopic and roentgenologic examination.

In infancy, collection of the urine presents a problem which is only solved by the care and ingenuity of the nurse. In the male, a test tube fixed to the penis with a finger-cot or adhesive tape affords a convenient method of collection while in the female, the application of specially fitting glass or rubber appliances which can be held in place by the diaper are utilized. For accurate diagnosis in females, a catheterized specimen should be secured after the labia have been separated and the perimeatal area thoroughly cleansed with boric acid or an antiseptic solution as bichloride of mercury (1 to 1000) or oxymercure of mercury (1 to 1000). In the male, the glans can be thoroughly cleansed with an antiseptic solution with the prepuce retracted. The first urine voided is discarded and the balance saved. This simple procedure avoids the need of

catheterization. The latter is easily performed, however, and should not be avoided if indicated.

Having obtained a urine specimen, the urologist is chiefly interested in its macroscopic appearance, reaction, microscopic cytologic findings, and the presence or absence of bacteria as evidenced by direct smear and culture on suitable media.

*Macroscopically*, even a clear urine does not rule out underlying pathology. In tuberculosis a clear lemon yellow urine is a characteristic finding together with a sterile pyuria. Nor is a hazy specimen necessarily significant. Addition of a few drops of 6 per cent acetic acid may produce a rapid clearing of the urine, either with effervescence due to carbonates or without it (phosphates). Regulation of the diet will frequently correct this common but harmless condition. The failure of the urine to clear points to the existence of pyuria. This can be determined by addition of sodium or potassium hydroxide with a resultant gelatinous precipitate, final confirmation being made microscopically.

*Gross hematuria* usually presents no problem in recognition. However, one must not be confused by abnormally colored urine due to ingestion of artificially colored candies or foods (beets, rhubarb) or the administration of drugs (indigo-carmin, methylene blue, pyridium).

We have found the routine racking of a patient's urine an exceedingly helpful and informative procedure. A series of test tubes appropriately labeled are set up in a rack and represent a portion of each voided specimen. The degree of pyuria or hematuria and its progression or regression under observation or treatment can be roughly evaluated in this way.

The reaction of the urine assumes a certain degree of importance in dealing with urinary infections and lithiasis, both as a pre- and postoperative problem. It has been shown that bacterial growth is either inhibited or destroyed at a pH of 5.5 or less. This is especially true of the *B. coli* infections. The role of the hydrogen ion concentration in urinary tract infections and urolithiasis is presented at greater length under the sections dealing with these disease entities.

Various dyes have been used as indicators in determining the pH of the urine. Methyl red, the universal indicator, special commercial colorimeters, and chlorphenol red are particularly suitable because of their applicability to the pH range in which urologists are interested. A simple technique which we have found useful in the office and at the bedside is the addition of 1 or 2 drops of 0.01 per cent chlorphenol red to about 20 drops of urine in a test tube. Whereas the indicator itself is orange in color below a pH of 5.6, a yellow color is produced and progressively above it, on the alkaline side, a faint pink color develops into a deep violet red hue at an

above a pH of 6.8. The method is simple and rapid and gives valuable information as to the bacteriostatic properties of the urine. This is especially true when one is using a drug such as methenamine which is valueless unless the urine is sufficiently acid to liberate formaldehyde.

The specific gravity of the urine concerns the urologist only as it reflects the functional capacity of the kidneys to carry on especially under any surgical procedure. A fixed specific gravity is a grave omen and surgery should be deferred or undertaken with caution since a bilateral nephritic lesion may exist.

Microscopically the outstanding pathologic findings of importance to the urologist are (1) Pus cells (2) red blood cells (3) bacteria and (4) tumor cells. The finding of casts should of course not be dismissed without further clinical investigation.

The sediment obtained from centrifuging the specimen should be examined carefully first under low and then under high power magnification. Finding more than 6 to 8 pus cells per high power field in a catheterized specimen is abnormal. Similarly the occurrence of red blood cells where trauma due to instrumentation can be excluded should always merit further investigation. Motile bacteria may be visible on the slide under the microscope and fixing and staining of the sediment using either methylene blue or preferably Gram stain may provide further information. In over 90 per cent of cases Gram positive cocci will be found to be either staphylococci or streptococci and Gram negative bacilli will belong to the colon group.

Occasionally in cases suspected of neoplastic disease a diagnosis has been made by finding tumor cells in the centrifugized urine either from the bladder or as obtained from either ureter through a catheter. Fragments of villi vacuolated hypernephroma cells or large irregular multinuclear cells may prove to be pathognomonic. The sediment obtained from centrifuging equal parts of the urine specimen and 20 per cent formalin should be fixed run through alcohol embedded in paraffin and section and staining carried out as for the usual pathologic specimen.

**Bacteriologic Study** The first requisite is care in obtaining the urine specimen under strict aseptic technique. Secondly and something that is overlooked is the necessity for starting the laboratory investigation as soon after the specimen is obtained as possible. Too often the urine is allowed to stand until the close of the day and casually deposited in the laboratory along with routine specimens. As a result a prolific organism may completely overgrow the real bacterial offender.

Six organisms—the B. colon, staphylococcus, streptococcus, gonococcus, tubercle bacillus and *Proteus vulgaris* account for over 90 per cent of all urinary tract infections. These and their sub-

groups can be readily identified by suitable staining and cultural technic. We would stress the importance of routinely investigating every pyuria for tubercle bacilli by careful repeated studies of direct smears or if necessary, by guinea pig inoculations. The finding of sterile pyuria is strong evidence for tuberculous etiology.

Outstanding among the abnormal urinary findings seen by the urologist are pyuria and hematuria. Because of their frequency and importance they will be dealt with at some length.

**Pyuria**—Pyuria is not a disease. It merely reflects the presence of a focus of infection in the genito-urinary tract. The term pyelitis is frequently used to cover the presence of pus in the urine. It too is a misnomer. To clarify the approach to determining the etiology of a pyuria we shall consider the subject under seven broad headings.

1 *Infections*—The causal agents are usually the coli or cocci. Clinically and pathologically, a pyelonephritis, pyelocystitis or pyonephrosis is present. The lesion is almost always secondary to a portal of entry in the nasopharynx, upper respiratory tract, middle ear or intestinal tract, by the hematogenous route. Ascending infections and those conveyed by lymphatic extension account for a small group of cases.

2 *Malformations*—Anomalies either congenital or acquired may produce in turn obstruction, stasis and infection. Among such lesions are stenosis of the prepuce, stenosis of the external meatus, urethral stricture, urethral diverticulum, posterior urethral valves, hypertrophy of the verumontanum, ureteral stricture, ureteral diverticulum, aberrant vessels with ureteral obstruction, hydro-ureter and polycystic kidney disease.

3 *Lithiasis*—The incidence of stones either in the kidney, ureter, bladder or urethra may be the forerunner of infection. Irritation of the mucosa presents a point of lowered resistance for bacterial invasion. Frequently stones produce a mechanical obstruction causing stasis and subsequent infection. On the other hand stones may result from infection, inspissated pus acting as a nidus and altered chemical equilibrium causing precipitation of crystals out of solution.

4 *Tuberculosis*—Pyuria may be the only manifestation of a tuberculous lesion in the genito-urinary tract. Pus in the absence of bacteria, so-called sterile pyuria—strongly suggests tuberculosis.

5 *Neurogenic Lesions*—The atonicity resulting from dysfunction of the nerve innervation to the ureter, bladder or vesical sphincters results in stasis and infection. Primary spinal cord pathology or lesions secondary to vertebral anomalies such as spina bifida account for the majority of cases.

6 *Neoplasms*—By producing obstruction by degenerating or by becoming a site of infection tumors often produce a pyuria.



7 *Extra-urinary Causes* —Periureteral or perinephritic suppuration may occur subsequent to vertebral caries, psoas abscess, ovarian or tubal abscess, appendiceal or intestinal foci, or suppuration in the prostate or seminal vesicles

In making a diagnosis of pyuria, it is essential that the physician knows how the specimen of urine was obtained. To this end, the external genitalia should be carefully inspected for urethritis or vaginitis. In such cases, a catheterized specimen is obligatory. Clear urine does not necessarily rule out suppuration in the urinary tract, for the focus may be walled off completely or may discharge intermittently. High temperatures also may temporarily cause suppression of discharge. Occasionally, what appears to be clear urine suddenly becomes turbid when the last part of the specimen is obtained, due to the pus gravitating and making its presence evident in the residual urine.

Having made a diagnosis of pyuria, one should not temporize. If it continues in spite of medical treatment of *four to six weeks*, a complete urologic investigation should be undertaken. The finding represents tangible evidence of trouble and accounts for 80 per cent of pediatric urologic cases.

**Hematuria** —Being only a symptom, hematuria demands prompt attention in determining its source. There has been a tendency to procrastination on the part of the practitioner in seeking the aid of the urologist in such cases. Too often medication is prescribed and the patient told to return if the blood reappears. Not infrequently there is no recurrence for some time but the underlying process is still present and progressing. Hematuria represents a danger signal and the opportune moment for urologic consultation is during the active period of bleeding. At that time the exact point of bleeding can often be determined cystoscopically, either by direct visualization, or indirectly by catheterizing the ureters.

In the following outline, the numerous causes of hematuria will be presented. The multiplicity of sources attests to the utter hopelessness of attempting accurate diagnosis without exacting study of each case.

#### I *Systemic or Extra-urinary Causes*

A *Diseases of the Hematopoietic System* —Hemophilia, polycythemia, purpura hemorrhagica, symptomatic purpura, paroxysmal hematuria, leukemia, Hodgkin's disease. Scurvy, while metabolic in origin, causes bleeding as a result of altered capillary permeability.

B *Infections* —Toxic nephritis due to bacterial infections in septicemia, subacute bacterial endocarditis, typhoid, diphtheria, meningitis, dysentery, influenza, smallpox and scarlet fever.

I *Systemic or Extra-urinary Causes* — (Continued)

C *Lesions of Adjacent Organs* — Appendicitis, perinephritis and pelvic abscess. The mechanism in appendicitis is produced by the pelvic type with perimetritis by contiguity.

D *Medicinal or Toxic* — Hexamethylenamine, turpentine, cantharides, mercury, phosphorus and lead.

II *Urogenital Causes*

A *Renal* (While any disease of the kidney can cause hematuria, the important entities follow.)

- 1 Glomerulonephritis
- 2 Infections—pyogenic
- 3 Tuberculosis
- 4 Tumors including polycystic disease
- 5 Calculi
- 6 Trauma

B *Ureteral*

- 1 Infections—ureteritis.
- 2 Stricture
- 3 Calculi
- 4 Tumors or anomalies
- 5 Trauma

C *Vesical*

- 1 Infections—acute or chronic cystitis
- 2 Foreign bodies
- 3 Calculi
- 4 Tumors
- 5 Trauma

D *Urethral*

- 1 Infections—urethritis
- 2 Ulceration of the meatus or prepuce
- 3 Stricture
- 4 Foreign bodies
- 5 Trauma

**Renal Function Tests** — Before undertaking any surgery, one must be certain of the capacity of each kidney to carry on its work. This is especially important when nephrectomy is contemplated. While kidney function tests are resorted to more extensively in dealing with medical nephritides, the surgeon relies upon them as a qualitative measure of operability and risk, and to indicate repair and recovery postoperatively.

The two outstanding means of determining kidney function are

- 1 Dye excretion
- 2 Blood chemistry

Such tests are not infallible nor do they give us a really accurate picture of what is going on in the kidney proper. They merely

represent the functional status of the organ at the time of the test. Nevertheless from experience the data made available has proven to be a reasonably accurate guide and for this reason both tests should always be carried out as part of a complete urologic study.

The excretory powers of the kidney are usually measured by using either phenolsulphonaphthalein or indigo carmine. We are not unmindful of the value of concentration and dilution urea clearance urea ratio and kindred determinations. These are used to better advantage in chronic nephritics.

Indigo carmine marketed in 5 cc ampoules of an 0.8 per cent concentration of the dye is perhaps the most convenient agent for determining the qualitative status of the kidneys. Practically all cases in which a definite urologic lesion is suspected come to cystoscopy. At that time 0.5 cc of the drug is injected into one of the intercubital veins. Rarely in infants it may be necessary to use a more accessible vein of the scalp, ankle or neck (external jugular).

Normally indigo carmine makes its appearance at the ureteral orifices in four to eight minutes. The dye escapes from the meris in increasing intensity until a deep blue color is attained. For practical purposes the concentration is set down as 1, 2, 3 or 4+. This method of mentoscopy is not fool proof however. Total excretion of fluid from the kidneys may be so diminished by preoperative dehydration as to fail to provide a vehicle for the injected dye. Again the dye may be completely decolorized or greatly diluted and lead to misinterpretation. Occasionally excretory inhibition, ureteral spasm or actual obstruction will interfere with appearance of the dye. In such cases the passage of an ureteral catheter results in prompt appearance of the dye often with a continuous flow of urine from the tube indicative of retention due to obstruction.

In all doubtful cases a re-check should be done either with the same dye or a different one. In cases that present any difficulties in identification of normal landmarks during cystoscopy, indigo carmine often proves invaluable. The visible ejection of the dye aids in locating the ureteral orifices which may otherwise be obscured by edema, tuberculous or neoplastic infiltration.

Phenolsulphonaphthalein was first popularized by Rowntree and Geraghty and represents the most accurate means at our disposal for determining kidney function by a dyestuff. It is frequently referred to as the PSP or phthalein test. The dye is available in 1 cc ampoules containing 0.6 mg of the monosodium phenol sulphonaphthalein in solution. It may be given intramuscularly (intralumbar, intragluteal or intradeltoid) or intravenously. The latter route is accurate and finds its best use at the time of cystoscopy, ureteral catheterization and collection of specimens.

Given intravenously the dye appears in two to five minutes normally and 90 to 95 per cent appears from each side within the first

fifteen minutes and 5 to 10 per cent in the second fifteen minutes. This is equivalent to a total of 50 to 70 per cent in the first half hour. For practical purposes test tubes containing an alkali can be used as receptacles for the catheter drainage. Alkalinization is necessary to bring out the pink to red color of the dye and a suitable set of standards or a colorimeter provides ready means for percentage determinations. In addition to the factors previously mentioned is interfering with the appearance of indigo carmine. Phthalein concentration may be lowered by leakage about the ureteral catheter. To offset this the bladder urine should be saved and measured to correct the gross error. As the 100 per cent standard is 1 cc PSP in 1000 cc of water it is necessary to so dilute all specimens collected for a proper percentage reading.

If the intramuscular route is utilized excretion is naturally slower and the question of rapidity of absorption enters into the accuracy of the test. Two glasses of water should be given prior to the injection to promote urinary excretion. Normally the percentage output of the phenolsulphonephthalein in the first hour is 40 to 60 per cent and 15 to 25 per cent in the second hour. In children collection is more accurately carried out by catheterizing, discarding the specimen obtained when the dye is first injected and collecting at half hour intervals with the catheter indwelling in the urethra. If a catheter is not used the bladder is emptied after the injection and specimens voided at half hour intervals are saved for two hours. The difficulty here lies in failure of the child to cooperate.

Both indigo-carmin and phthalein have their greatest applicability in chronic cases. Whereas in acute cases there may be no evidence of any impairment in chronic lesions diminished PSP function precedes any blood chemistry changes. Both tests find utility not only in diagnosis but as an indication of progress or cure as evidenced by subsequent repetitions of the renal function tests.

Blood chemistry studies are especially informative in denoting retention of nitrogenous waste products normally excreted by the kidneys. We shall deal with the essential blood constituents as they concern the urologist. The following table gives normal blood chemistry findings in children.

Non protein nitrogen (NPN)	20 to 35 mg per 100 cc
Urea nitrogen (urea N)	10 to 15 m <sup>m</sup> per 100 cc
Ureae d	2 to 3.5 mg per 100 cc
Creatinine	1 to 2 m <sup>m</sup> per 100 cc
Sugar	80 to 100 mg per 100 cc
Chlorides (NaCl)	450 to 500 mg per 100 cc
Carbon dioxide combining power	45 to 65 volumes per cent
Calcium	9.5 to 11.5 m <sup>m</sup> per 100 cc
Phosphorus	4 to 6 m <sup>m</sup> per 100 cc

With marked kidney destruction the above constituents tend to accumulate in the blood thereby producing the condition known

as azotemia, or nitrogen retention. It is surprising, at times to find advanced surgical lesions in one or both kidneys without any appreciable alteration in the blood chemistry of the individual. The remarkable ability of but a small portion of a kidney to carry on the work of both kidneys, riddled with disease, represents one of the marvels of the human structure. In obstructive uropathies, advanced nitrogen retention occurs and relief of the obstruction either by temporary mechanical means (catheter) or corrective surgery (cystostomy, nephrostomy) often results in a remarkable clearing of the azotemic condition. In this connection it has been frequently stated that a creatinine over 6 mg means an early demise. Urologists frequently see ambulatory cases with values up to 25 mg. These represent cases with indirect renal parenchymal involvement on an obstructive basis, and of long duration.

**Co<sub>2</sub> Combining Power** — Determination of the carbon dioxide combining power of the blood serum is especially important in children whose acid base equilibrium is subject to rapid alteration. This liability is a constant source of worry to the pediatrician as well as to the urologist because of the rapidity with which a sick child can go into a state of alkalosis or acidosis during any extended period of vomiting or diarrhea. Severe degrees of acidosis are frequently encountered in acute or chronic infections of the kidneys.

For practical purposes, we depend upon the urea nitrogen sugar, and carbon dioxide combining power determinations. The urea nitrogen is a reliable indicator of the excretory power of the kidneys. If it is elevated, non-protein nitrogen and creatinine readings are made. Calcium and phosphorus values are significant in such metabolic lesions as renal rickets and as possible etiologic factors in calculus formation. A high phosphate appears to parallel nitrogen retention. In the interpretation of all these readings it is important to correlate them with the clinical picture. Thus, with dehydration, high nitrogen values may be due to the oliguria and not to any intrinsic renal pathology. We have not mentioned albumin-globulin ratios, lipid or cholesterol determinations because they belong in the realm of the clinician and pediatrician.

**Instrumentation** — *The child who comes to the attention of the urologist for specific study is a sensitized individual. He or she has probably been examined with some degree of discomfort and been stuck with at least one needle. The vision of a bougie catheter or cystoscope is not welcomed whole-heartedly. In fact in infants and children who cannot be made to understand it is usually the occasion for lusty crying and great physical activity. For the simple procedures, as probing or catheterization the infant must be forcibly restrained by the nurse. In older children, fair cooperation can be obtained.*

Cystoscopy and ureteral catheterization in infants and the majority of young children is best carried out under general anesthesia.

The urethra represents the path which must be traversed in all mid- and upper urinary tract investigations. For this purpose, a variety of agents are available. Urethral instrumentation includes the determination of the patency of the canal and the existence and location of any obstructing tissues. Urethral catheterization represents the simplest means at hand. The instrument of choice is a small soft rubber catheter of the Nélaton type, with blunt tip and lateral eye. The size may vary from 6 F to 18 F, in the case of older girls. Parenthetically it might be stated that size gradation is usually expressed in terms of the French unit, which is equivalent to 0.3 mm. The diameter of a 6 F catheter is 2 mm, while that of an 18 F instrument is 6 mm.

No difficulty is encountered in females, but in males the small-sized soft rubber catheter may buckle, and with a child straining and bearing down, its passage is difficult, if not impossible. Accordingly, a comparably-sized woven silk urethral catheter or a large size (10 F) ureteral catheter will provide the necessary rigidity to permit passage of the hollow tube into the bladder. The use of rigid instruments such as steel sounds should be avoided since considerable trauma may result, especially in a struggling child.

If a point of obstruction is encountered in the urethra, or if one wishes to explore anomalous orifices or passages, suitable whalebone filiforms, or woven silk whips with hollow followers (Phillips or LePort type) are available. Graduated woven silk bougies or steel natural curve sounds find only occasional use in the pediatric examination.

Needless to say, strict asepsis is carried out in all such procedures. The instruments are sterilized either by boiling soft rubber or steel articles, while woven silk instruments are kept immersed in mercuric oxyvanade 1 to 1000, or kept dry in a formalin cabinet.

**Urethral Catheterization**—The procedure serves a number of purposes. It represents a means of exploration, of obtaining sterile bladder urine specimens, of ascertaining residual urine, of determining bladder capacity, and finally in providing a means of carrying out cystometric observations as well as cystography. The actual technique while simple, merits description. The examiner should either wear sterile gloves or render his hands surgically clean by scrubbing. A sterile water-soluble lubricant, of which many are on the market, or simple nitrobenzene or glycerin, may be used.

A sterile towel above and one below the field of operation is desirable. The catheter, lubricant, cotton balls and antiseptic solution, and sterile receptacle should be within easy reach of the physician. In the female, the nurse serves best by holding the child with the lower extremities flexed at the knees and hips.

abducted. The labia are separated and drawn upward and outward by the thumb and index finger of one hand while the perineal region and external meatus are swabbed from above downward with a cotton pledget saturated with green soap or mercury oxyvanide 1 to 5000. The catheter is then picked up at two points, the distal end and 1 inch from the proximal eye. In its introduction into the urethra the tube is thus more easily handled and contamination is avoided.

In the male the same general technic is followed. The prepuce is retracted and the glans and meatus are cleansed as the penis is held between the thumb and index or middle finger with the lips of the external meatus separated. The catheter is introduced slowly and without force. A sense of resistance is encountered when the external sphincter is reached but this is readily overcome and the posterior urethra traversed with ease.

We have presented the above procedure in detail because we feel it is not too elementary to be considered. Catheterization has frequently been rendered unduly difficult through the inaptitude of the inexperienced. Apart from the absence of finesse is the more important possibility of adding insult to injury by the introduction of infection through careless or faulty technic.

**Cystometry**—This finds a limited application in the examination of the child. It is based on the normal response of the bladder musculature to varying degrees of distention. With a catheter in place a sterile solution is allowed to enter the bladder either by gravity or pressure depending upon the method used. Rose, Hyams, Muschat and others have devised special apparatus for such determinations. The examiner notes water volume and pressure at the first desire to void, at the sensation of fullness and at the time of pain of overdistention. Suitable curves or kymographic records may be made for permanent recording.

In the adult the first desire to void is usually at 150 cc with an intravesical pressure of 8 to 10 mm. fullness attains at 250 cc and the pain of overdistention at 350 cc. These values are lower for children but have not been as clearly studied or formulated. The value of the test lies in differentiating the true obstructive bladder lesion from the atonic neurogenic lesion without obstruction.

**Cystoscopy**—Cystoscopy of the infant and child at the present time is a far cry from the days of its inception. The original instruments introduced by Nitze and Portner over thirty years ago were unwieldy, observation cystoscopes being of the same length as the adult type. The very small diameter of the tube resulted in bending which in turn impaired vision. In 1911 Edwin Beer devised a shorter instrument, size 10 F for observation and 15 F for catheterization of the ureters. With the advent of this and subsequent cystoscopes (McCarthy, Young, Butterfield, Corliss

and Campbell), urologic investigation of the child entered upon a new era of progress. Cystoscopy has been done on infants in the first month of life.

The construction and relative merits of the various miniature cystoscopes are readily obtainable from commercial catalogues. The individual urologist should possess the dexterity and the instruments which in his hands have been most useful. We have found the McCarthy foroblique pan-endoscope useful in practically all cases. In the very young the McCarthy miniature cystoscope embodying the foroblique visual system, with 14 F, 13 F and 11 F sheaths provide in turn for double catheterization, single catheterization and simple observation.

The female urethra will accommodate sizes from 16 F to 21 F easily. It must be emphasized that the limitation of the field of vision of the miniature cystoscopes may lead to error. For this reason we favor a general anesthetic if necessary, to enable the passage of equally large size instruments in the male to carry out cystoscopy, bilateral ureteral catheterization, and operative procedures such as cutting or coagulation with the electric loop or electrode.

*Introduction of the Cystoscope*—In experienced hands this is relatively simple and painless. Rarely is a meatotomy necessary. No anesthetic topical application caudal or general anesthesia may be necessary. The examiner must choose to the best interests of his patient. The greatest obstacle to successful cystoscopy without general anesthesia is overcoming the child's fear and apprehension in the bewildering environment of instruments, roentgen ray tubes and darkness. The eradication of apprehension and physical struggle with its attendant danger of serious trauma afforded by administration of a general anesthetic far outweighs the objection to such narcosis.

*The Cystoscopic Examination*—A definite routine should be carried out for cystoscopy. The child should be made to empty the bladder completely before introducing the instrument. Having done this the following are recorded:

- 1 Measurement of residual urine
- 2 Bladder capacity determination by water distention
- 3 Visual inspection of the bladder trigone, ureteral orifices, vesical neck and posterior urethra
- 4 Ureteral catheterization
  - (a) Sterile specimens for bacteriologic study
  - (b) Pyelography

In obstructive lesions—whether organic or neurogenic—residual urine is an index to the degree of dysfunction. In children one may be misled by the failure of the child to fully empty the bladder. The test should be repeated at a later date using a soft rubber catheter. Having recorded the absence or presence of residual urine



and noting whether the bladder medium contains flakes of mucus the viscous is lavaged thoroughly. All modern cystoscopes have inflow and outflow tubes in contrast to the antiquated observation instruments which depended upon preliminary filling of the bladder before introduction of the instrument.

With localized ulceration or diffuse cystitis bladder tolerance to distention is poor. Having distended the bladder the presence of a foreign body, calculus or tumor is excluded. The vesical mucosa is carefully inspected and any deviation from the normally pale pink surface noted. The child's bladder lies higher than the adult's and its relative intra-abdominal position causes it to move with normal respiratory excursions of the viscera.

Next the trigonal area is inspected and the ureteral orifices are located. They are relatively large in the child and readily admit a 5 F or 6 F ureteral catheter. Before introducing catheters however the presence or absence of urinary flow from each orifice is noted. We favor obtaining sterile ureteral specimens at this point and then administering indigo carmine to test kidney function. In this way the urine is uncolored by the dye and hematuria or pyuria is recognized immediately without being masked by coloration of the dye stuff. The dye tests using either indigo carmine or phenolsulphonephthalein have been described previously.

**Ureteral Catheterization**—In passing catheters up each ureter any point of apparent or real obstruction should be noted. The specimens obtained from the renal pelvis are sent to the laboratory for microscopic culture and B. tuberculosis study. When the catheter reaches the renal pelvis and a continual flow of urine is obtained it should be collected, measured and the degree of pyelocaliectasis determined. With the catheters *in situ* retrograde pyelography can be performed as indicated.

At the completion of the investigation of the upper urinary tract and as the cystoscope is withdrawn the foroblique vision embodied in the pan endoscope affords a perfect inspection of the urethra in both males and females. The presence of diverticula, posterior urethral valves, hypertrophy of the verumontanum or neoplasm is thus determined. Reactions following cystoscopy are surprisingly few. Campbell (1931) noted a 12 per cent temperature elevation after 293 examinations. Fluids should be forced and an urinary antiseptic prescribed. Methenamine and sodium acid phosphate in doses of 15 grains each three daily are effectual. There should be no hesitation in raising this dosage to 75 grains daily if the condition warrants it. Codein sulphate grain  $\frac{1}{4}$  will help control any pain.

In disease the above-outlined routine for cystoscopic investigation may be abandoned or altered to suit the individual case. Discovering a pyonephrotic lesion by intravenous urography should discourage one from passing catheters indiscriminately up the op-

posite ureter. The injected dye may appear in diminished concentration or not at all suggestive but not proof positive of renal pathology. In cases of hematuria the optimum time for cystoscopy is at the time of bleeding so that the exact source may be localized. Blood in the ureteral specimen must be properly evaluated since the trauma occasioned by passage of the catheter may be the cause. One could continue indefinitely with the possibilities that may manifest themselves at the cystoscopic table. A thorough basic knowledge of urology augmented by the proper armamentarium and the ability to use such with sound judgment embody the main attributes of successful diagnosis and therapy.

**Urography**—The combined efforts of the chemist and clinician in developing suitable drugs for visualization of the genito-urinary tract through the medium of roentgen ray represents the final epochal contribution to the field of diagnostic urology. The names of von Lichtenberg, Binz and Swick will always be associated with the introduction of uroselectan, the drug which really opened the field of intravenous urography.

The starting point in radiographic diagnosis of the genito-urinary tract disease is the so-called diagnostic flat plate of the abdomen. It is also referred to as the preliminary or K U B (kidney ureter bladder) exposure. From it one can ascertain the presence or absence of any soft tissue or skeletal abnormalities, shadows of increased density suggestive of calculi and the position and contour of the kidneys.

The question of preliminary preparation of the gastro-intestinal tract has been a subject of lively discussion. Without elaborating unduly, several facts stand out. Children are prone to greater gas accumulation than adults. They are also subject to greater reaction to drastic catharsis. In a child whose alimentary canal has been well regulated, no preparation may frequently produce as satisfactory pictures as if an ounce of castor oil the night before and a cleansing enema the morning of the roentgen ray appointment had been administered.

Others feel that dehydrating the youngster is the best and only means of procuring ideal plates. This is effected by withholding all foods and fluids the evening prior and the morning of the examination. This is readily applicable to diagnostic and intravenous urography, but when cystoscopy is undertaken in combination with roentgen ray investigation, the examiner will find kidney function greatly diminished, obtaining of specimens slow and arduous and dye excretion tests delayed by the diminution in fluid vehicle.

In sum, one cannot generalize or indict because satisfactory results are obtained by those who favor strong catharsis and starvation as well as by those who omit any preparation. For intravenous urography, our routine has been 1 ounce of castor oil at 7 P.M. the eve before the examination after a light 3 P.M. supper. Nothing is

given by mouth thereafter until after the roentgen ray examination which is scheduled for 9 A M the following morning For retrograde pyelography catharsis is frequently omitted a regular supper is given and fluids permitted until an hour before the cystoscopy In this way dehydration that will interfere with proper kidney function and specimen collection is avoided and the direct instillation of the contrast medium in more concentrated form counteracts any gaseous distention

The routes for administration of radiopaque substances are (1) Intravenous (2) by retrograde pyelography (3) subcutaneous and (4) oral

Intravenous or excretory urography has attained a merited prominence in the past five years The affinity of the kidneys for certain complex iodine preparations has been made capital of by research workers and pharmaceutical organizations Popular preparations are neo-skiodan or diodrast (3,5 diiodo-4 pyridon \ acetic acid diethanolamine) skiodan (mono-iodo-methane sulphonate of sodium) and uro-selectan or neo-iopax (disodium salt of 3,5 diiodo-4 pyridoxyl \ methyl 2,6 dicarboxylic acid) These are administered intravenously in 10 to 20 cc dosage depending on the age and size of the child Administration is simple but care should be taken to inject slowly and avoid extravasation of the solution outside the vein

While intravenous urography is a simple and relatively harmless procedure its shortcomings as well as its advantages must be considered As a preliminary source of information it is invaluable Where cystoscopy and ureteral catheterization is unwarranted undesirable impossible or even refused it is an excellent means of providing a clue to the underlying pathology However in a series of 60 case studies Schwentker (1932) found the method a diagnostic help in 60 per cent of 42 children over two and in only 30 per cent of 14 cases under two years of age Campbell in a series of 304 cases found intravenous urography unsatisfactory in one-third under the most favorable conditions

Administration of the drug is theoretically contraindicated in cases of severe renal or hepatic insufficiency acute tuberculosis and the exudative diatheses Practically however it has been used in many such cases without sequelae

Interpretation of roentgen ray plates secured by intravenous urography should not be final They may lull one into a sense of security whereas retrograde studies may uncover real pathology Similarly incomplete filling of minor calyces may simulate frank pathology only to find normally delineated pelves after retrograde pyelography In short while intravenous urography can easily be carried on by the pediatrician or the roentgenologist he would be failing in his duty if he were not to consult with the urologist Further the urologist who depends solely on excretory urographic findings for undertaking surgery fails to serve the child's best interests

Intravenous urography may well become a routine step in the urologic investigation of children since valuable clues may be afforded that will guide the urologist in his subsequent instrumental examination. In addition it provides a rough estimate of kidney function in the majority of cases.

**Retrograde Pyelography** Retrograde pyelography represents the introduction of a radiopaque substance into the pelvis and ureter by means of ureteral catheters at the time of cystoscopy. The substances used for intravenous urography are also used for this procedure and being well tolerated by the blood stream are safest and least irritating to the urinary tract mucosa. However the dictates of economy lead many to use other halogen preparations which are perfectly safe. These include sodium iodide and hippuran a newer preparation which we use routinely at the present time.

The value of retrograde pyelography lies in its usual clearer definition and more accurate information on the extent of tissue damage as evidenced by dye replacement. At the same time one is afforded an opportunity of carrying on phenolsulphonephthalein or indigo-carmin function tests and collecting specimens for culture. Where the latter is the prime reason for the work up one may dispense with intravenous radiography entirely in favor of cystoscopy specimen collections and retrograde pyelography thus saving time and money. A point in technic bears emphasis. Whenever possible a uteropyelogram should be made and adequate distention determined by ureteral reflux of the dye observed through the cystoscope. The placement of catheters to the renal pelvis and injection of the contrast medium blindly may obscure lesions at the uretero-pelvic juncture or in the ureter.

**Cystography and Urethrocytography** must be mentioned in the consideration of roentgen ray investigation of the lower urinary tract. Filling the bladder and urethral canal with contrast medium affords valuable information on the status of this region. Hematuria and acute infection are the only real contraindications. Deformities due to chronic infection, diverticulum or neuromuscular or organic vesical neck obstruction may be clearly demonstrated by the method (Fig. 229). The procedure is carried out by filling the bladder through a catheter with a 5 per cent solution of sodium iodide. If delineation of the urethra is desired the catheter is removed and the urethral canal is distended by injecting a 70 per cent hippuran solution *via* the external meatus. In older children the bladder should be filled until the first desire to void manifests itself. In the young and inarticulate 1 ounce of the iodide solution is usually sufficient for a two-year-old while 2 or 3 ounces may be used up to the age of six years.

**Subcutaneous and oral administration** is a preliminary to excretory urography is now in the process of development. It may well be

in general usage in the very near future. Its weakness lies in the uncertainty of its assimilation and the time factor in taking roentgen ray exposures. The majority of contributions have come from abroad. Butzengeiger (1931) reported the successful administration of a 4 per cent skiodan solution (20 grams in 500 cc. of water) in adults. Hildebrand (1932) injected 100 cc. of a similar solution into the axillary space of a child aged fourteen months with a renal tumor. Intravenous injection was reported as being impossible. The child died after the nephrectomy but autopsy revealed no significant or untoward changes at the site of the injection.



Fig. 229.—Chronically distended bladder with beginning irregularity and oval contour in an 8-year-old girl with vesical neck constriction. (Courtesy of Dr. S. R. Woodruff.)

Hildebrand (1932) and Nissil (1932) used abrodil and perabrodil respectively as a subcutaneous injection for excretory urography. Beer and Theodore (1934) employed 7 per cent neo-skiodan in 50 cc. injections in 10 children. No untoward reactions occurred and satisfactory urograms were taken thirty to ninety minutes after the injection.

Oral administration has been attempted by Swick (1933). He used a preparation of 10 to 15 grams of sodium orthoiodohippurate dissolved in simple syrup. The drug has a salty aromatic taste and produced no nausea, vomiting or diarrhea. Films not quite as clear in definition as by the intravenous route were taken at sixty, ninety, one hundred and twenty and one hundred and fifty minute intervals.

## CHAPTER XXXIX

### UROLOGIC SURGERY IN THE CHILD

THE indications for surgery in dealing with pediatric urologic conditions differ in no wise from the same problems in adults. Campbell (1931) in reviewing a series of juvenile cases found the following reasons for investigation: pyuria 80 per cent, disturbed urination 15 per cent, hematuria 2 per cent, tumor 2 per cent and pain 1 per cent.

The discovery of surgical pathology as a result of study of the above enumerated subjective or objective findings demands prompt attention. Too often is a policy of observation carried on. Preservation or conservation of the genito-urinary apparatus is essential for the future well being of the child. Localized suppuration must be drained early and adequately. There is no reason for complete renal destruction when a loin incision and drainage will relieve perinephric suppuration. The causes of obstruction to the flow of urine, whether due to stone, aberrant vessel, congenital stricture, diverticulum or tumor, warrant prompt surgical intervention as do disabling anomalies, tumors or specific disease entities.

**Anesthesia** —Because of the age of our subjects and the natural fear and apprehension in the hospital atmosphere, the subject of anesthesia is a vital one. Certain urologists favor the omission of general anesthetics and as little local anesthesia as is conveniently possible. In urethral, vesical and ureteral instrumentation, as well as during urography, it would be a decided advantage to have the cooperation of the patient. However, it often is questionable whether the possible danger of self injury by a struggling or suddenly fear-stricken child can compensate for the omission of narcosis.

The types of anesthetic agents at our disposal are: inhalation, topical, local, regional, spinal, intravenous and rectal.

**Inhalation Agents** —Ether has been for many years the anesthetic of choice in pediatric urology. Its ease of administration and its margin of safety enhances its value. The use of ethyl chloride or chloroform by open drop is fraught with danger and is not advised, although some experts in their use still employ them to advantage. In older children, nitrous oxide, ethylene or cyclopropane may be used, often to greater advantage for operations of short duration than can be attained with ether.

**Topical Anesthesia** —Topical anesthesia in the form of applications, instillations or injections of agents over the urethral or vesical

mucosa are useful preliminary to cystoscopic instrumentation in older girls less often in boys. Five per cent cocaine on a cotton applicator makes a very satisfactory agent for topical application to the external meatus and distal  $\frac{1}{2}$  inch of the urethra. For urethra and bladder one of the many procaine derivatives issued under various trade names may be used. We have found a 1 per cent solution of diothane efficacious using 1 ounce in the bladder and urethra and also using it on the meatal applicator.

**Local Infiltration**—Local infiltration finds limited usage. Novocaine in 1 to 2 per cent solution is used in older children for minor surgery. Circumcision can be performed very satisfactorily with it in cooperative boys ten years old or over.

**Regional Anesthesia**—Regional anesthesia by field block is seldom used in children. It may however supplement a basal anesthetic such as avertin. Nerve block as utilized in the procedure of *sacral* or *caudal anesthesia* is finding greater applicability and usefulness in the hands of some urologists. It has been used for cystoscopic examinations, urethral and perineal operations (urethrotomy, fulguration of papillomata and vesical neck surgery). Its usage is confined to males since the ease of instrumentation in females does not warrant it.

**Technic of Caudal Anesthesia**—This is not difficult. With the patient lying on his abdomen and the pelvis elevated by a small pillow the lower sacral area is prepared and draped. The sacral notch is localized by palpation of the two sacral cornua and the depression between them represents the location of the sacro-coccygeal membrane which is to be penetrated. Having had roentgen ray studies previously one can gain additional information as to the bony conformation by a study of the preliminary plate. A thick panniculus often renders clear definition of the space difficult.

A wheal is raised over the site with 1 per cent novocaine and a 22 gauge needle  $1\frac{1}{2}$  inch in length is used to penetrate the skin, fat, fascia and membrane to enter the sacral canal. Before injecting 8 to 10 cc. of a 2 per cent novocaine solution aspirate with a dry syringe to assure the absence of blood or spinal fluid. Occasionally the needle may penetrate a blood vessel or rarely the meningocele of a spina bifida or unusual extension of the subarachnoid space may yield spinal fluid. The ease with which the anesthetic solution is injected and the absence of subcutaneous infiltration are further criteria of successful injection.

For a fair trial the operator should wait twenty minutes after the administration before beginning any instrumentation. If successful the anesthetic is an ideal one. However one must realize that the child is fearful of any procedure in an operating room environment—he may become unruly or hysterical apart from any infliction of pain. Under such circumstances the examination

proves to be a very trying procedure for all concerned. Furthermore even in expert hands crural anesthesia is not uniformly successful. Campbell (1933) reported 80 per cent success in 83 cases with 8.4 per cent partially satisfactory and 10.8 failures.

**Spinal Anesthesia.** Spinal anesthesia has been used by some in children over twelve years of age. We do not advocate its use in juveniles feeling that other means at our disposal are much more effective and safer.

**Intravenous Anesthesia.**—Intravenous anesthesia has had a cyclic popularity. A few years ago Zerfas and McCallum (1929) reported success with sodium amtal administered intravenously. Subsequent adverse reports impaired its further popularization. In the last three years a new barbiturate, evipal, has met with considerable favor. Chemically it is N-methyl-cyclo-hexenyl methyl malonyl urea which was isolated by Kropp and Traub (1932) in the course of experiments on barbituric acid derivatives. Over 50,000 injections of evipal as it is known abroad have been given in various European countries notably Germany and England. The Medical Research Council of the Royal Society of Medicine in London (1933) reported 1 death in 25,000 cases.

The sodium salt of the drug is used for anesthesia. It is a water soluble powder supplied in 1 gm ampoules and dissolved in sterile triple distilled water to make a 10 per cent solution. We have modified this technic using a larger bulk of water as a vehicle thus avoiding any clot formation in the needle since the drug is injected very slowly and in as small a dosage as will keep the patient asleep and relaxed. Some make a single injection of 0.6 gm and proceed. We do not favor this method.

The value of evipal lies in its ease of administration, the rapidity of onset of anesthesia with complete relaxation, the wide margin of safety and the promptness of recovery without disagreeable aftermath. The patient who is engaged in conversation during the administration of the drug gradually lags in his speech and goes into a deep sleep without any period of excitement or discomfort. Its danger lies in respiratory failure but avoidance of overdose will safeguard against such an occurrence. One should have made all preparations for the operation prior to giving the drug since maximum anesthesia is attained using ordinary dosages in twenty to forty seconds. We have used it successfully for circumcisions, cystoscopic examinations, meatotomies, incision and drainages, excision of hydroceles and other minor urologic surgical procedures.

**Rectal Anesthesia.**—Rectal anesthesia has been assuming a position of importance in pediatric surgery since the introduction of avertin fluid or tribromethanol amylenehydrate. By its use one is enabled to avoid the struggle, shock and fearful memories in the juvenile subject. The drug, a white crystalline substance, is dis-



pensed as avertin fluid which is dissolved in distilled water at 40° C. It is given slowly through a rectal tube while the patient is in bed the child thinking an enema is being given. For children the dosage varies between 80 and 100 mg. per kilogram body weight. A suitable table is supplied with the drug to facilitate accurate preparation. Failures or accidents have been ascribed to improper dosage and administration.

Since avertin acting as a basal anesthetic produces a period of narcosis varying from one and a half to two hours it is usually restricted to operations of some duration in short major urologic surgery. Children will be found to be more tolerant to avertin so that the adult dosage table can be followed. The basal anesthesia produced is often not as deep as procured in adults so that it may be found necessary to employ supplemental inhalation or local anesthesia.

We have presented a rather sketchy outline of anesthetic agents as they apply to pediatric candidates for urologic surgery. In evaluating the agents at our disposal we feel that by and large inhalation anesthesia is still the anesthetic of choice. Caudal intravenous and rectal anesthetic agents all have their place and in experienced hands yield gratifying results. Concerning the question of omission of anesthesia other than topical application for cystoscopy, ureteral catheterization and pyelography when adults approach the procedure with trepidation how can we hope for such an ideal in children in their initial exposure to white gowns, the darkened cystoscopic room and overhead roentgen ray apparatus? Full cooperation on their part is rare indeed and rather than subject them to undue shock and possible injury we feel it far better that most of them be oblivious of their surgical surroundings through the medium of a general anesthetic. An anesthetic which leaves the child in full possession of his faculties of perception should be preceded by administration of a sedative. The habituates have been found useful in this connection.

**Prognosis**—Unless the child is the victim of long standing irreparable renal damage the immediate and remote prognosis following surgical intervention is better than that of the adult. Children as a group manifest unusual recuperative powers to any form of surgery and the field of urology is no exception. Complications may arise due to their greater susceptibility to intercurrent upper respiratory infections, otitis media and the erythremia.

The two groups of cases in which ultimate prognosis is bad are those of malignancy and chronic obstructive uropathy. Malignancy in the child is especially vicious and even with the use of roentgen ray therapy to supplement surgery the outlook is gloomy. What appears to be a relatively benign tumor which shells out with the greatest of ease at operation may manifest itself within the

proves to be a very trying procedure for all concerned. Furthermore even in expert hands caudal anesthesia is not uniformly successful. Campbell (1933) reported 80 per cent success in 83 cases with 8.4 per cent partially satisfactory and 10.8 failures.

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The two groups of cases in which ultimate prognosis is bad are those of malignancy and chronic obstructive uropathy. Malignancy in the child is especially vicious and even with the use of roentgen ray therapy to supplement surgery, the outlook is gloomy. What appears to be a relatively benign tumor which shells out with the greatest of ease at operation may manifest itself within the

year by metastases in the lungs. The rate of growth, rapidity of recurrence and metastasis are especially marked in childhood. The renal insufficiency produced by an obstruction to the outflow of urine with subsequent damming back and pressure atrophy often proves an insurmountable hurdle for its victim. Unfortunately, congenital obstructions at the vesical neck due either to organic or neurogenic causes often pass unnoticed until the child is in an uremic state. Then even though the obstruction be removed the damage wrought is often insufficient to preserve life especially should infection either local or systemic supervene.

Tuberculosis in the child is characterized by a favorable immediate prognosis but poor remote outcome. The younger the child the greater the difficulty of coping with the infection. While most children who are the victims of tuberculosis are subject to the miliary type the few who do localize in the kidney face the possibility of miliary dissemination at any time.

Surgery on the genital organs is attended with the lowest mortality when the urinary tract is not a factor. Where the urinary apparatus is linked with genital anomaly and requires transposition the added factor of infection must be considered. Statistics on the various surgical lesions will be presented as each is considered in the following pages.

## CHAPTER XL

### UROLOGIC DISEASES

#### ANOMALIES

WHEN one realizes that 40 per cent of all malformations of the body are confined to the genito-urinary tract (Schippers and Lange 1978) the significance of early recognition of such conditions in the child is apparent. Thus in a series of 4903 autopsies on infants Bugbee and Wollstein (1924) found anomalies in 104 or 2.3 per cent of the cases. Hyman (1976) reporting on a series of 150 children with major urologic problems reveals one third to have their etiologic origin in anomalies. Agam Campbell (1936) finds a 30 per cent incidence of malformations in 580 cases of chronic pyuria. 179 showing 206 anomalies of the upper urinary tract alone.

One could recount further statistical evidence of the unusual incidence of urinary and genital tract congenital defects. Their frequency and the difficulties they offer to accurate diagnosis are readily acknowledged. We are more concerned with them however as they affect the future well being of their host. Many of the anomalies especially those of the upper urinary tract may be carried well into adult life without apparent symptomatology. In contrast deformities of the external genitalia are readily apparent. Ironically enough such lesions which have little effect on the longevity of the child are manifest and cause considerable parental agitation whereas the much more important congenital defects with associated obstructive uropathies are carried unnoticed for extended periods of time with insidious progressive destruction of life preserving tissue.

Except in the rare case where serious defects are accidentally discovered anomalies usually manifest themselves clinically in disturbances of urination, pyuria (stasis and obstruction) or by uremia. A large mass may be noted in the lower hypogastrium or pyuria may persist over a period of years despite a rigorous medical therapeutic régime. Genital malformations are manifested by sexual dysfunction or anticipation of such in the child by the parent.

Surgery is indicated necessary and often a life saving measure in combating the effects of urinary tract anomalies. Because of the innate recuperative ability of children conservation by plastic surgical technique is resorted to whenever possible. The extent of the sacrifice of tissue by the surgeon's scalpel parallels delay in diagnosis and

treatment. It is remarkable how kidneys which to all outward appearances are mere shells will respond to surgical intervention when an obstructive uropathy exists.

The explanation of the creation of anomalies lies in faulty embryologic development. The comparative anatomy and development of the urogenital organs make an interesting if somewhat complicated study. It is beyond the scope of this treatise to attempt any detailed discussion of the complex subject.

**Embryology.** This will be limited to a brief exposition of basic facts. Urogenital development follows four fundamentals (Hinman).

1 The urogenital sinus, genital tubercle and phallus form the external genitalia, the lower urinary tract and the accessory sexual glands by urinary and genital association. Malformations of the penis, scrotum, urethra, prostate, Cowper's glands and homologous female genitalia and bladder fall into this category.

2 The gonad and primary excretory duct form the testis and its efferent ducts by the urogenital union. Faulty union results in anomalies of the male and female genital tracts. In the male the epididymis, vas deferens, seminal vesicles, ejaculatory ducts; in the female homologous persistence defects occur. Agenesis in the urinary apparatus not infrequently is associated in the male with partial or total absence of the seminal tract while in the female as many as one-half may show double vagina, uterus unicornus or bifidus, absence of the uterus, Fallopian tubes, ovaries or vagina.

3 The Mullerian duct, a female fundment, degenerates in the male. The primary excretory duct is largely a male fundment and degenerates in the female. This interplay between male and female variants of the Mullerian and primary excretory ducts is a frequent source of sexual maldevelopment.

4 The ureteral bud and nephrogenic blastema form the permanent ureter, pelvis and kidney. Anomalies, some innocuous, others detrimental to life, result from embryologic dysfunction in this group. The specific incidence, diagnosis and treatment of individual anomalies in the urogenital tract will be discussed in ensuing sections as each organ is considered in detail.

## THE OBSTRUCTIVE UROPATHY

Because of the high incidence of congenital abnormalities in children, the problem of mechanical interference with the normal egress of urine becomes highly significant. Any obstruction whatever its cause results in stasis and subsequent back pressure as the trapped fluid increases in volume. Accompanying stasis is the ever-present hazard of infection and the triad of obstruction, stasis and infection represents one of the most serious hazards with which its victim and his physician have to cope.

**Etiology**—The causes of obstruction or stasis are numerous. For purposes of brevity the significant ones will be listed as they apply to the child.

In the urethra congenital lesions include atresia stricture (usually meatal) diverticulum posterior urethral valves and hypertrophied verumontanum. Acquired conditions are stricture urethrocele calculus neoplasm and diverticulum. Phimosis and paraphimosis either congenital or acquired afford indirect causes of urethral obstruction.

In the bladder contracture of the neck with hyperplastic folds of vesical mucosa cysts and diverticulum are congenital occurrences. Acquired conditions include the above listed anomalies in addition to calculus neoplasm and vesical and sphincteric dysfunction due to peripheral or central nervous system derangements.

The ureter may be the site of congenital lesions such as valves folds strictures ureterocele and constriction by aberrant vessels. Calculus neoplasm stricture angulation and infection are acquired lesions causing obstruction. Congenital causes of renal obstruction are linked with anomalies of the pelvis aberrant renal vessels crossing and constricting the pelvic outlet and abnormal positions of the kidney. Calculus neoplasm or nephroptosis account for acquired obstructive pathology.

While obstruction usually antedates infection the sequence may be reversed. An initial infection may result in sufficient local tissue reaction with attendant edema infiltration scarring or stricture formation to produce an impaired urinary channel. Such a process may eventuate from recurrent attacks of pyelonephritis or from such specific entities as tuberculosis or gonorrhea.

**Extra urinary Factors** Obstruction to the outflow of urine is caused by several extra urinary factors. We have already alluded to the dynamic stasis produced by neurogenic lesions. Its significance lies chiefly in disturbed innervation to the bladder and its sphincter less frequently in a large atonic ureteral lesion. Pelvic masses arising from either anomaly infection or new growth in adjacent organs may produce mechanical obstruction by pressure. Belt (1936) reported a case of massive hematocolpos behind an imperforate hymen in a child suffering from incontinence of urine. The bladder neck and urethra were so encroached upon as to produce a condition of paradoxical incontinence the dribbling being the overflow from a chronically distended bladder.

**Trauma**—Occasionally obstruction may develop subsequent to local damage incurred by trauma. The insult may be direct or indirect and may be due to external violence or to faulty instrumentation or operative intervention that does not take into account the possibility of disability at the site at a later date. Specifically merely cutting through a strictured area and opening the lumen of

a hollow viscus adequately will result in even greater secondary stricture formation unless a regular and diligent course of dilatation is carried on postoperatively.

**Results of Obstruction**—The pathologic results of long-standing unrelieved obstruction are dependent on principles of hydrodynamics. The urinary apparatus may be considered to be a tubular channel as represented in the ureters and urethra. Nature very thoughtfully incorporated buffers or reservoirs, the renal pelvis and bladder, into the system. De-



FIG. 230—Advanced pyonephrosis and pyoureter due to obstruction by a vesical diverticulum in a boy aged ten years. Uretero-nephrectomy was performed.



FIG. 231—Incised surgical specimen of same child—the kidney merely a shell.

spite these safeguards, the hydrostatic pressure produced by continued secretion of urine behind a complete or partial obstruction cannot be long denied without extensive tissue damage.

Because the urinary tract is normally a continuous passage from



glomerulus to external meatus an obstruction at any point in the path will result in changes above that site. Thus a lesion of the urethra can involve in turn the bladder ureter and kidneys. Obstruction at the uretero pelvic junction involves the pelvis and renal substance.

Obstruction irrespective of the cause produces certain definite changes in the segments of the urinary tract. Urethral obstruction is characterized by dilatation. Infection or congenital defect may so weaken the urethral wall as to cause diverticulum formation or even extravasation. In the bladder hypertrophy trabeculation cellule formation diverticulum and finally dilatation result. Hypertrophy of the detrusor mechanism and interureteral ridge is produced by the attempts to expel the entrapped vesical contents.

The ureter whether it be the primary seat of the obstruction or indirectly involved may show hypertrophy or dilatation with associated elongation angulation and loop formation with adhesions. The renal pelvis undergoes hypertrophy and dilatation and the kidney suffers primary atrophy of simple hydronephrosis or secondary atrophy of infection. The final stage with infection sooner or later being superimposed is represented by pyonephrosis (figs 230 and 231.)

**Clinical Manifestations**—Obstruction at or below the vesical neck manifests itself by some form of disturbed urination. Complete atresia of the urethra of course permits no passage of urine and is incompatible with life if unrelieved. Partial obstruction is characterized by diminution in the urinary stream straining or dribbling in paradoxical incontinence. The lesions of the ureter and kidney unfortunately progress insidiously until a mass appears or are otherwise discovered by pyuria systemic and febrile sequelae of infection or uremic manifestations.

**Treatment of Obstructive Lesions**—This involves removal of the obstruction either by conservative plastic repair or sacrifice of the destroyed organ. Prognosis depends upon the degree of tissue destruction and the factor of infection. The majority of children so affected reach the urologist in far advanced stages of disease. The entire subject of incidence treatment and prognosis will be dilated upon under respective sections dealing with specific organ pathology.

## PYOGENIC INFECTIONS

We have elected to consider infections of the genito urinary tract in general because such conditions usually involve more than one organ. Furthermore only by so dealing with urogenital infections can one hope to clarify the rather confused phases of etiology and pathology. We shall however deal specifically with infections as

diseases of the respective organs constituting the tract are presented in succeeding pages

**Etiology** The origins of urinary tract infections are diverse. Sporadic cases have been reported of its incidence in newborn babies. During the early creative years and before the child has developed any degree of immunity to bacterial invasion it may be a prey for such infections and the urinary tract may frequently be secondarily affected. Finally the introduction of foreign bodies, injuries or faulty instrumentation have accounted for a few cases of infection.

Although a surgeon is consulted only when a suppurative focus requires drainage or when the lesion causing the infection is to be eradicated, he must be fully aware of the nature of the causative agent, the pathology created and the most suitable lines of therapy to be used concurrently with his surgical endeavor.

Malnutrition or food deficiencies ranging from starvation to avitaminosis may so devitalize the child as to produce ideal conditions for the bacteria already harbored in the body. A study by Bloch (1931) showed systemic and localized infections in 80 per cent of a group of 32 scorbutic children. The urinary tract was a frequent site of the infection in both groups. In vitamin A deficiency Tyson and Smith (1929) and Killian and Grewal (1935) noted epithelial degeneration in the urinary tract with numerous abscesses.

On the other hand the well nourished child is often the victim of acute pyelonephritis. The primary focus in such instances is usually found in the ear, nasopharynx, pulmonary or gastro-intestinal tracts. The preponderance of colon infections has led to a careful study of the relationship of the gastro-intestinal and urinary tracts. Schwartz found Gram negative bacilli in 60 per cent and definite bacilluria in 21 per cent of the urines of children with gastro-intestinal diseases.

**Causative Organisms** The etiology of urinary tract infections evolves from the offending agent and the route of infection. A detailed discussion of pyuria has already been presented (Page 626). The principal organisms causing such infections are

#### A. *Bacilli*

##### I. Colon typhoid group

1. *B. coli communior*
2. *B. lactis aerogenes*
3. *B. typhosus*
4. *B. paratyphosus* A and B
5. *B. dysenteriae*

##### II. *B. proteus*

##### III. *B. pyocyaneus*

##### IV. *B. fecalis alkaligenes*

##### V. *B. mucosus capsulatus* (Friedländer's bacillus)

B *Cocci*

- I *Staphylococcus* (albus and aureus)
- II *Streptococcus* (hemolytic non hemolytic and viridans)
- III *Micrococcus* *cristallalis*
- IV *Pneumococcus*

The colon group of organisms accounts for over 80 per cent of all urinary tract infections in children. Some believe that cocci may represent the initial infective agents with the colon bacillus as a secondary invader and overgrowing all other organisms. At least 10 per cent of the remaining cases are due to the action of the staphylococci.

Specific agents which produce characteristic lesions not included in the above groups and which will be considered under organ pathology are

- 1 *Tubercle bacillus*
- 2 *Gonococcus*
- 3 *Actinomyces*
- 4 *Spirocheta pallida*
- 5 *Amœbe*
- 6 *Echinococcus*
- 7 *Schistosoma*

The routes of invasion of bacteria into the genito urinary tract are three

- 1 Hematogenous or endogenous
- 2 Ascending or urogenitogenous
- 3 Lymphogenous

Without entering into any extended discussion the hematogenous route of infection is most frequently encountered in children. The source may be specific systemic infections or fevers (septicemia, bacteremia, typhoid, scarlet fever, dysentery) or focal points of origin may lie in the skin (furuncles), bones (osteomyelitis), infected teeth, tonsils, adenoids, middle ear, upper respiratory or intestinal infections.

The ascending route is less common but accounts for a certain number of cases in females. Herrold (1922) has shown that colon bacilli from the urine and cervix were identical serologically and biologically. The lymphogenous route may account for a few cases. Some dispute the point but definite lymphatic channels have been demonstrated between the colon and upper urinary tract.

Closely allied with the determination of the route of infection is the problem of ascertaining whether a particular infection falls into the spontaneous or complicating groups of Hellstrom (1924). The latter types in contradistinction to the former have intrinsic pathologic lesions as causative factors. While calculi, trauma or specific lesions like tuberculosis account for a few such cases the majority fall into the group of obstructive renal infection. Obstruc-

tive uropathy and the various congenital and acquired conditions which may contribute to its creation have already been considered

**Pathology** The usual finding is that of a pyelonephritis with or without ureteritis and cystitis. The term pyelitis so commonly used is an obvious misnomer. Such an infection without complication will undergo rather quick resolution under ordinary medical care. Chronicity of infection is an indication of probable organic pathology as obstruction complicating the picture. The result may be suppurative nephritis, infected hydronephrosis, pyonephrosis, renal carbuncle or perirenal suppuration. As a rule staphylococcus infection tends to localize and accounts for the greater number of such suppurative processes.

With an hematogenous infection and an initial cortical focus the pathologic changes are reversed. Here the infection proceeds downward with the evacuation of the pus into the pelvis, ureter and bladder. In this connection finding a clear urine in the face of loin tenderness, fever or pyelographic evidence of renal suppuration should be discounted since the pus may be walled off.

**Symptomatology**—Infection in the urinary tract may be characterized by fever, continuous or remittent, gastro-intestinal symptoms (anorexia, diarrhea, vomiting, tympanites), pallor, urinary disturbances (frequency, nocturia, dysuria, pyuria or hematuria).

Infections of the genitalia in children are relatively infrequent if one were to exclude the pyogenic lesions of the external genitals due to uncleanness. The numerous infantile infectious diseases may occasionally manifest a focus in this region as the orchitis or oophoritis of mumps. These will be considered later in the presentation of organ pathology.

**Treatment** Treatment of urinary tract infections will be considered in detail at this point since the routine is applicable to handling an infection in any of the organs constituting the system. It will also obviate subsequent unnecessary repetition. The foundation upon which effective therapy rests is early and accurate determination of the cause of the infection. Simple pyelonephritis which does not respond to the usual medical routine of rest, forcing fluids, bowel hygiene and urinary antiseptics and pyuria which persists longer than one month demand prompt urologic investigation. The relative merits and application of complete urinalysis, bacteriologic study, roentgen ray, cystoscopy and ureteral catheterization have already been presented.

The subject will be considered under the following headings:

- 1 General measures: rest, fluid regulation, bowel hygiene
- 2 Urinary antiseptics
- 3 Regulation of the reaction of the urine
- 4 Diet
- 5 Bacteriophage

- 6 Elimination of extra urinary foci
- 7 Instrumentation
- 8 Surgical intervention

For the acute infection or one complicating instrumentation important measures are bed rest and the forcing of fluids. Water has been aptly termed the best urinary antiseptic at our disposal. However when one is prescribing an urinary antiseptic it is well to realize that the fluid intake should be restricted in order to enhance the effectivity of the drug. This is a point frequently overlooked in one's zeal to force fluids. Again the only accurate means of knowing the patient's fluid water balance is to accurately record on the bedside chart the total intake and output of fluids.

Because the function of the gastro intestinal tract is so closely related to that of the urinary apparatus care should be taken to see that its normal regulation is unimpaired. Catharsis should be effected both by medication as well as by mechanical cleansing of the lower bowel. For this purpose a daily enema or periodic colonic irrigations are indicated and form an integral part of the therapeutic program.

**Urinary Antiseptics**—Urinary antiseptics administered orally are numerous and varied. In our hands hexamethylenamine has been used to greater advantage in combination with an acidulating salt such as acid sodium phosphate or ammonium chloride. The important factor in their use is prescribing large enough doses. Children tolerate the drugs exceptionally well. At least 40 to 60 grains of each drug should be given daily. A flexible rule of 10 grains per year of age may be followed so that a child aged five years would receive 50 grains of methenamine and a like amount of the acidulating medium. It is essential that the urine be acid for effective use of methenamine so as to promote the liberation of the antiseptic formaldehyde radical in the urine. The pH of the urine can be rapidly determined by dye indicators. (See section on Examination of Urine.)

Other drugs have found favor in the hands of some urologists. Hexylresorcinol (Caprokol), acriflavine and pyridium find strong advocates and equally strong denouncers. Edwin Davis (1932) in a careful study of the drugs as determined by antiseptic tests of samples of urine obtained before and after their administration in unit maximum doses to normal persons concluded that pyridium is practically inert, caprokol is slightly antiseptic, methenamine is quite efficient and acriflavine (in alkaline urine) is unfailing. He also found that acriflavine administered as shellac-coated pills was inert while its administration in ordinary capsules though non-injurious caused unpleasant symptoms of nausea and catharsis.

The advent of the ketogenic diet led to closer investigation of the basic reason for its ability to render the urine sterile in certain

cases. It was found that the two agents responsible were the lowering of the pH of the urine below 5.5 and the presence of beta hydroxy butyric acid in the urine in increased amounts. Each was enhanced by the other in its efficacy. An attempt to administer beta hydroxy butyric acid by mouth resulted in failure because the substance was oxidized before it reached the kidneys.

Mandelic acid was found to be an aromatic acid with definite bacteriostatic powers and a worthy substitute for the above-mentioned ketone body by Ro-enheim (1935) of the University College Hospital London. Several American workers (Helmholz and Osterberg 1936, Cook and Buchtel 1936, Dolan 1936) have reported generally satisfactory results especially for the *Escherichia coli* group of organisms.

The drug is administered as mandelic acid in divided doses totaling 10 to 12 gm daily together with adequate amounts of acidulating salts to produce a pH of 5.5 or less. For children we have found the commercially prepared syrups combining both the mandelic acid and ammonium chloride most effective and palatable. Two drams of syrup which represents a 40 per cent solution of mandelic acid are prescribed four times daily. It must be remembered that the drug is still in its experimental stage and wider application will either prove its merit, bring out its limitations or consign it to the oblivion of numerous other drugs that had a meteoric existence. Several cases of induced hematuria have already been reported. The necessity for using an acidifying agent may produce some degree of gastric upset and it is always important to remember that the child can easily be forced into an acidotic state by indiscriminate use of such agents.

**Intravenous Medication.**—The intravenous route of administering urinary antiseptics offers a more direct and rapid attack in severe cases of infection. Methenamine in its various commercial forms (urotropin, uretone and amphomate) can be given in varying doses from 30 to 60 grains at a time and repeated if necessary. At least one commercial preparation (amphomate) contains both methenamine and sufficient camphoric acid to provide the necessary acidulation. Hugh Young has long advocated mercurochrome by the intravenous route. For the fulminating case of staphylococcus infection we have used neoparsphenamine intravenously with gratifying results in doses of 0.3 gm every other day. Good results are usually obtained in seven to ten days.

**Reaction of the Urine.**—This in itself is of assistance in the treatment of urinary tract infections. Shohl and Janney (1917) showed that colon bacilli were retarded in their growth in a urine whose hydrogen ion concentration was 5 or less on the acid side or 9.2 or more on the alkaline side. On this principle various acids or alkali producing drugs have found their applicability. Acid sodium

phosphate or benzoate ammonium chloride or nitrate and hydrochloric or nitrohydrochloric acid have been administered

To produce an alkaline reaction sodium bicarbonate acetate or citrate alone or in combination are given in daily doses sufficient to attain the desired pH. This may require as high as 80 to 100 grains of the drug daily. In large or long-continued acid or alkali medication the need for guarding against the production of an alkalosis or ketosis cannot be too strongly emphasized.

**Balsamic Medication**—For controlling the vesical or urethral irritation that so frequently accompanies infective processes we have found balsamics either alone or in combination with acid or alkaline therapy of considerable value. Sandalwood oil or cubebs 10 minims each or tincture of hyoseyamus 30 minims four times daily usually allays tenesmus and burning.

**Importance of Diet**—The importance of the diet of a child ill with urinary tract infection has often been slighted. In acute pyelonephritides the traditional treatment has included forcing of water and fruit juices plus alkaline medication. The dietetic management depends upon the severity of the reaction to the infection. A suitable menu can be selected from such easily digested and nourishing foods as orange juice, milk, stewed fruits, gruels with cream, soft boiled or poached eggs, toast or bread, butter, preserves, rice, tapioca and custard. As clinical improvement occurs, tender meats and easily digested vegetables may be added to the dietary.

**Ketogenic Diet**—In chronic cases the ketogenic diet has found a certain degree of popularity since its introduction by Helmbolz and Clark (1931). The former (1935) reported on a group of 21 cases with anomalies, stasis and infection with temporary cures in 5 and permanent relief in 6 of the children.

The basis of ketosis production lies in the restriction of carbohydrates with consequent arrested fat metabolism and the production of ketone bodies (acetone, diacetic and beta hydroxybutyric acids) in the urine. The diet while best administered in the hospital can be made available for office or out patient use. Carbohydrates are limited to 15 to 25 gm., proteins to maintenance levels of 0.3 gm. per pound of normal body weight and fats in sufficient quantities to meet caloric requirements of the body.

Clark and Keltz (1935) have attained adequate ketosis with a diet composed of 1½ pints of 40 per cent cream and 6 eggs any style in twenty four hours. In carrying out any dietary regime the fulfilment of all the nutritional requirements of the growing child must be accomplished. Adequate caloric intake including sufficient calcium, phosphorus, iron and vitamins is essential. Because of this dictum and the difficulty of juvenile cooperation, other methods of combating infection are to be preferred to the ketogenic diet.

**Alkaline Ash Diet**—This has a restricted application in certain types of infection with or without stone formation. It is antagonistic to the precipitation of cystine, oxalic or uric acid calculi. Meats are best omitted and all vegetables with the exception of corn should be given liberally.

**Acid Ash Diet**—The diet is used either alone or with acid medication in stubborn suppurative infections with or without pyelopathic lithiasis. All meats, poultry, fish and cheese are permitted while most vegetables are excluded.

For a complete presentation of the subject of dietetics in pediatric urology, including sample diets, indications, rationale and application, the reader is referred to the authors' section in Saxl's *Pediatric Dietetics* (1937).

**Bacteriophage Therapy**—This has been a valuable adjunct to treating certain otherwise resistant urinary tract infections. Several definite conclusions appear justifiable:

1. To be effective, the agent must either be a specific one or a stock phage which demonstrates a high degree of lysis toward the offending organism. The indiscriminate application of stock or commercial preparations without exact bacteriologic studies is bound to be fruitless.

2. Bacteriophage should be used in adequate dosage by every route available. Subcutaneous or intravenous injection should be combined with bladder or renal pelvic instillation. A suppurative postoperative wound may be treated by wet dressings or instillation of phage directly into its depths.

3. For maximum efficiency of the bacteriophage, the pH of the urine should be close to 7, since acid urines inhibit lysis of bacteria. This can be attained by administering alkalis orally.

Conflicting reports on the value of bacteriophage stud the literature. Larkum (1927) noted susceptible organisms in only 40 per cent of the urines examined by him and 36 per cent contained bacteriophage. MacNeal, Frisbee and Applebaum (1934) in analyzing a group of 97 patients treated, found 23 cured clinically and bacteriologically, 27 with symptomatic improvement with final bacteriologic proof of eradication of the infection, 23 failures and 24 with inadequate data as to the result. Krueger, Faber and Schultz (1930) from a carefully studied group of 89 children with urinary tract infections, express considerable doubt as to how much value could be attributed to the bacteriophage *per se*.

Our own feeling in the matter is that nothing is lost and much can be gained by giving a potent bacteriophage. Much depends upon the skill of the bacteriologist in this respect and it has been our good fortune to have associated with us in this work Dr. MacNeal and his co-workers who have made notable contributions in the



field. Only by close cooperation between clinician, bacteriologist and patient can maximum benefit be derived from bacteriophage therapy.

The elimination of foci elsewhere in the body contributing directly or indirectly to the onset or persistence of the infection is essential. Acute or chronic lesions of the ear, oropharynx or teeth should receive specialistic attention.

**Instrumental Intervention**—This is indicated at any stage in the treatment of a case of urinary infection. While usually contraindicated in the acute phase, judicious use of the urethral or ureteral catheter for facilitating drainage is often imperative. In his armamentarium the urologist has diverse means for promoting drainage from or treating directly the site of infection.

**Urethral catheterization** may be performed repeatedly or the catheter left indwelling to aid drainage or to permit lavage with such antiseptics as 4 per cent boric acid solution, 1 to 3500 potassium permanganate, 1 to 3000 neutral acriflavine, 1 to 2500 metaphen solution or bacteriophage. Cystoscopy may be used with or without ureteral catheterization to relieve any temporary obstruction to evacuate pus or to permit the introduction of antiseptic solutions as 0.5 to 2 per cent silver nitrate or other commercial colloidal silver preparations, mercurochrome or bacteriophage. The ureteral catheter may be left indwelling in the ureter from twenty-four to seventy-two hours as a therapeutic aid in combating ureteral or renal infection.

**Surgical Treatment**—Surgical treatment is undertaken as indicated and will be dealt with under proper subject headings. Cystostomy, nephrostomy or perirenal drainage are standard procedures in selected cases. In the extreme case nephrectomy may be necessary and while outwardly it may appear to be a radical step it has often proven to be a life-saving measure. In concluding this section on the treatment of urinary tract infections we must reiterate that the aforesaid enumerated measures singly or in combination represent sound, rational prophylactic, palliative or therapeutic agents. Their use is warranted in:

1. Acute pyelonephritis or pyelocystitis
2. Chronic infections without demonstrable pathology as evidenced by cystoscopy and urography
3. Chronic infections with demonstrable pathology where operative therapy has failed or is contraindicated
4. Urinary infections developing postoperatively
5. Urinary infections following instrumentation with catheter, sounds or cystoscopes or as a prophylactic measure prior to cystoscopy

## TUBERCULOSIS OF THE UROGENITAL TRACT

**Incidence**—While *miliary tuberculosis* is common in the child the chronic or surgical lesion with which the urologist is concerned is relatively infrequent. Mathe (1936) in reviewing 4698 cases of unilateral surgical renal tuberculosis in all age groups found 267 or 12 per cent in children. However only 20 or 0.42 per cent occurred in children from one to five years of age with 51 or 1.08 per cent in the five to ten year age group. Combined we find a total incidence of 1.5 per cent in children up to ten years of age.

With each advancing year the incidence of renal tuberculosis rises perceptibly. In the ten to twenty year range 494 cases were found representing 10.5 per cent of Mathe's entire series. Kretschmer (1936) studied 43 cases of surgical renal tuberculosis and found 4 between the ages of four and ten years, 11 between the ages of eleven and fifteen, and 28 between sixteen and twenty-one years of age. Statistical studies in sex predilection show almost an equal distribution, the male victims slightly outnumbering the females.

The reason for the rarity of chronic tuberculosis in the child is due to the nature of the disease. Primary foci for the tubercle bacillus in the child are in the lungs, intestines or tonsils. The majority of such cases result from transmission of the organism from an adult with an open pulmonary lesion. With the advent of pasteurization or other bactericidal treatment of all except certified milk, the role of bovine type of tuberculosis has been a minor one. Theavian and bovine types generally produce lymph node or intestinal lesions primarily. Park and Krumwiede (1912) found bovine tubercle bacilli in 46 of 51 cases of gland tuberculosis. Such infections are rare in urogenital tuberculosis.

**The Primary Nidus**—The introduction of the tubercle bacillus into the pulmonary system is followed by the production of the primary complex consisting of the primary focus (Ghon's tubercle), tuberculous lymphangitis leading from this site to the regional lymph node and caseous lymphadenitis of this node. At any stage of the cycle dissemination or regression of the disease can occur. Extension may occur directly along lymphatics by rupturing into a bronchus, pleura, peritoneum or lastly by erosion into a blood vessel.

**Secondary Urologic Lesion**—The secondary lesion produced in the urinary or genital tract is usually blood borne. Rarely does the lymphogenous or ascending route account for the infection. Although the lungs usually represent the primary focus of the disease the culpable focus for the immediate urogenital lesion may be a secondary one elsewhere. A careful history and physical examination is therefore obligatory.

**History**—In the history one should inquire into antecedent pulmonary ailments. Pleurisy with effusion or empyema are significant when linked with definite parenchymal pathology. Cervical glands or residual scars should be noted. Ankylosis of a joint or a long standing suppurative lesion of a bone should draw one's attention to a tuberculous focus. Urinary symptoms especially frequency nocturia hematuria and pyuria with or without dysuria and urgency are highly significant. Kretschmer (1936) found an incidence of tuberculosis elsewhere in 72 per cent of a group of 43 cases of urogenital tuberculosis in children.

**Physical Examination**—This will often uncover pulmonary, bone joint or glandular tuberculous lesions which may have passed unnoticed. In the male child careful examination of the genitalia may reveal a tuberculous epididymitis, prostatitis or seminal vesiculitis.

Before considering pathology, symptomatology, diagnosis and treatment of urogenital tuberculosis one must be mindful of the fact that the kidney represents the initial focus of urinary tract infection while the epididymis, prostate and seminal vesicles are vulnerable points in the genital tract. The urinary and genital systems are so interwoven in form and function as to defy any attempts to consider them as separate entities in relation to tuberculous infection. Bumpus (1930) discovered renal involvement in 330 of 606 cases of genital tuberculosis in all age groups at the Mayo Clinic.

It is now almost universally agreed that an excretory bacilluria must leave its mark on kidney tissue. Medlar (1926) with Thomas and Kinsella (1927) showed the presence of microscopic tuberculous lesions in the renal parenchyma and also advanced strong evidence for considering most cases of renal tuberculosis as bilateral in origin.

The acute miliary lesion is characterized by generalized dissemination of the infection affecting both kidneys with a toxic tuberculous nephritis as represented by fibrosed, sclerotic organs. The pre-clinical lesion or stage of invasion with the finding of tubercle bacilli in the urine has led to the conflict of viewpoints on the subject of actual renal involvement as held by Medlar or an excretory bacilluria without local changes as taught by Wildbolz (1929).

The chronic surgical lesion of the urinary tract concerns us directly. Here again opinion varies as to whether healing takes place or whether the tuberculous process goes on slowly but progressively to eventual destruction of the organ. Microscopic tissue studies prove conclusively that healing does occur and the argument is really more academic than of clinical significance.

**Pathology**—Chronic tuberculosis in the genito-urinary tract as elsewhere is characterized by (1) tubercle formation (2) inflammatory reaction (3) caseation (4) nodule formation (5) fibrosis

and (6) calcification. One or more of these stages may be found present in the same organ.

**Renal Lesion**—The renal lesion may consist of caseocavernous destruction about a papilla or at the base of a pyramid with marked papillitis or abscess formation as represented in pyonephrosis. The kidney may take on the consistency of putty. Associated with the tuberculous lesion may be concomitant calculus, tumor or polycystic disease. Deposition of lime salts in areas of caseation gives rise to calcification producing the characteristic mottled shadows on the roentgen ray plate. The nodular tuberculous kidney is rarely seen. It represents a displacement of renal parenchyma by solid nodules of conglomerate tubercles which do not caseate. The fibrotic type of renal tuberculosis, while grossly similar to the nodular type is characterized microscopically by the absence of tubercles.

**Tuberculosis of the Ureter**—The ureter may be the resting place for tubercle bacilli carried down from the initial focus in the kidney. Implantation of the bacilli on the mucosa results in either a diffuse ureteritis or one localized to the lower end of the tubular structure.

The pathologic picture of ureteral tuberculosis depends upon the stage of the disease. One may find tubercles involving mucosa, muscularis and periureteral tissues with subsequent deformities, strictures and resultant dilatation. Caseation may result in the accumulation of necrotic detritus in the lumen of the ureter, which dilates above such a point. The ureteral wall may become greatly thickened, or canalized secondary to the caseous process. Contracture of the lower end of the ureter produces the characteristic golf hole ureteral orifice of advanced tuberculosis. Should autonephrectomy occur or the tuberculous focus above be removed surgically, the ureter usually involutes to become a cord like structure with obliteration of its lumen. The ureter, representing but a connective link in the urinary tract, cannot be properly considered as an entity but merely a part of the diffuse infection. Its involvement, however, may be the cause of pain or colic-like attacks.

**Tuberculosis of the Bladder**—When secondarily involved from renal tuberculosis the bladder shows either congestion, edema, ulceration or tubercle formation about the ureteral orifice. In the more advanced case the whole bladder may become involved in a diffuse inflammatory process with ulceration, purulent exudate, mucous membrane proliferation into polypoid masses or infiltration of the entire bladder. Subsequent vesical contracture results in intolerance, marked urgency and frequency. The ureteral orifices may by involvement and retraction of the lower ureteral segment produce typical golf hole type ureteral orifices.

When infection is genital in origin congestion and ulceration of the posterior urethra or vesical neck may be noted if urethroscopy is performed early in the disease.

**Symptomatology** — The symptomatology of tuberculous infection of the urinary tract is varied. Systemically one may note evidence of toxicity in subjective complaints of fever, cough, sweats, malaise, anorexia or recurrent gastro intestinal upsets. Intercurrent infections are common. Objectively pallor or loss of weight are outstanding findings. From an urologic viewpoint the outstanding symptoms are frequency, nocturia, pyuria, hematuria, burning and pain on urination, urgency or enuresis. Pain in the loin, constant or colicky, is a less common complaint but careful bimanual palpation may reveal a tumefaction in the renal area. The urinary symptoms are intensified by the tendency toward ulceration in tuberculous infection. Passage of an acid urine containing tuberculo toxins over such an inflamed surface together with the secondary contracture of the bladder makes the tenesmus, strangury, urgency and frequency of the victim of urinary tract tuberculosis readily understandable.

Kretschmer (1936) made some interesting observations on the duration of symptoms before diagnosis. The onset of symptoms in 17 of 43 juvenile cases (39.5 per cent) was less than six months, in 10 or 23.2 per cent from six months to one year, in 12 or 27.9 per cent from one to four years, and in 4 or 9.3 per cent from six to ten years. The answer to earlier diagnosis lies in constant vigilance on the part of the pediatrician entrusted with care of such children. Pyuria persisting for over four weeks under routine medical measures should be investigated forthwith from an urologic standpoint and careful urine studies made for tubercle bacilli. Recurrent pyelitis in addition to persistent pyuria has too often proven to be advanced tuberculosis. Such a benign condition as enuresis has upon ultimate investigation led to a diagnosis of renal tuberculosis.

**Diagnosis** — The diagnosis of genito urinary tract tuberculosis can be arrived at only by careful study of

1. History
2. Physical examination including renal palpation and examination of genital and prostate
3. Urinalysis
  - (a) The finding of tubercle bacilli in direct smear and stain of urinary sediment
  - (b) Sterile pyuria in an acid urine represents presumptive evidence
  - (c) Guinea pig inoculation
  - (d) Tuberculin as a therapeutic test — with intensification of urinary findings and symptoms

**History** — A background of familial tuberculosis or a record of loss of weight, malaise, cough, hemoptysis, enlarged cervical glands or bone disease or symptoms referable to the urinary tract may be disclosed. These may include disturbed urination, painless hema-

turia, dull loin pain or ureteral colics. On the other hand, a suggestive history may be entirely absent.

**Physical Examination**—This may reveal a phthisical appearance with pallor and loss of weight. Pulmonary, gland, bone or joint pathology may be noted. Examination of the genito-urinary tract may evidence tenderness in the loin or the presence of a mass. A tuberculous lesion of the genitalia, when discovered, should always be followed by a study of the urinary tract.



FIG. 232. Chronic renal tuberculosis in a boy, aged fifteen years. Note moth-eaten appearance of pelvic shadow.

**Kidney Function Tests**, utilizing phenol-sulphonephthalein or indigo carmine, may show normal excretion time, unilateral impairment, or complete loss of function. A final possibility is bilateral impairment of function.

**Urologic Roentgenology**—Urologic roentgenology should be preceded by an examination of the lungs where suspected, or of any suspicious bone or joint lesion. Frequently, however, while the presence of a Ghon tubercle or peribronchial thickening may be present, no evidence of an active renal lesion is noted.

The preliminary flat plate of the abdomen may show a large renal shadow or irregular calcific deposits in the renal area. Intra-

venous urography should always be done and may reveal characteristically moth eaten calyceal deformities pyonephrosis with islands of contrast medium separated from the pelvic shadow or complete excavation of the renal substance (Fig 232) The ureter and bladder may also show evidence of an ulcerative or fibrotic process

Retrograde pyelography should be undertaken only when the diagnosis is in doubt or to affirm a wavering decision as to the propriety of operative interference Caution in such instrumentation is necessary since the production of a generalized tuberculosis has been reported on several occasions subsequent to pyelography (Kearns 1927)

**Cystoscopic Examination**—Cystoscopic examination of the bladder may be essentially negative on inspection or may reveal varying degrees of involvement Hyperemic ulceration or actual tubercle formation may be visible about one or both ureteral orifices or may involve the whole bladder In this event a notable finding is marked bladder intolerance to instrumentation and distention so as to make general anesthesia mandatory for any satisfactory examination The ureteral orifices may be normal reddened edematous or retracted by ureteral involvement producing a golf hole type of deformity The urine ejected from each orifice may be clear bloody or frankly purulent Indigo carmine given intravenously at this point will reveal a fair evaluation of relative kidney function

Ureteral catheterization should be undertaken in all but the very obviously diagnosed cases taking every precaution not to introduce any new infection to a previously uninfected kidney Catheterization may be found difficult or impossible in the advanced case with ureteral fibrosis or obliteration The ureteral catheter may represent the deciding agent as to the unilaterality of the disease process by a study of the separated renal output

**Bacteriologic Studies**—With care and persistence the tubercle bacillus is demonstrable in about 90 per cent of cases The bladder urine should be studied for three successive days examining the sediment of collective twenty four hour specimens Urine obtained by ureteral catheterization is even more important in making a decision as to surgical intervention

A mixed infection with cocci or colon bacilli is a frequent occurrence and may mask the underlying tuberculous infection Guinea pig inoculation while not absolutely diagnostic due to animal immunity to certain strains is a reasonably reliable agent Lowenstein (1924) has found cultural methods even more exacting and has used the procedure routinely A 30 per cent sodium hydroxide solution or 40 per cent sulphuric acid is applied to the sediment to rid it of other organisms It is then washed three times and inoculated on glycerin potato or albuminous culture media

in children ureteral transplantation and cystectomy may be the only solution

**Results of Nephrectomy**—A composite study of nephrectomy for tuberculosis in several large clinics shows an immediate mortality of 25 per cent. Postoperative complications are few. Miliary tuberculosis involving the lungs or meninges may be set up by the operative intervention. Sinus formation at the operative site can be minimized by careful technic and while its persistence may prove annoying to patient and surgeon it usually closes without necessity for revision of the wound. Astringents or sclerotic agents may be used to promote healing but in most cases even these are of doubtful value. Bladder irritability may be treated postoperatively by lavage with antiseptic solutions (5 to 10 per cent argyrol 1 to 3000 potassium permanganate) with 1 to 400 phenol for its anesthetic effect or 25 per cent eucypt in oil or gomenol a proprietary vegetable oil. Such balsamics may also be used in the non-operative cases with similar vesical irritability.

**Prognosis of Renal Tuberculosis**—This is less favorable in children than in adults. Talc (1925) reported 24 per cent cures after nephrectomy in infants and children in Marion's Clinic. This compares with 56 per cent cures in adults. Far from being a radical step nephrectomy may be the most conservative one for the child. Stevens (1927) nephrectomized a thirteen months old child for renal tuberculosis.

Diminished constitutional resistance to tuberculosis in the young has increased the mortality rate. Diffuse miliary tuberculosis or involvement of the remaining kidney with eventual insufficiency and genital lesions has accounted for the majority of deaths.

**Genital Tuberculosis**—Tuberculosis of the genitalia is rare in the child. Its frequent association with urinary tract lesions has already been cited. Kretschmer (1936) noted 4 instances of tuberculous epididymitis in 43 cases of renal tuberculosis in children and adolescents. However none was under fifteen years of age.

**Primary Tuberculosis of the Penis** is extremely rare. The occurrence of a chronic ulceration and inguinal adenitis following ritual circumcision should be kept in mind. Wilson and Warthin (1912) collected 22 cases of tuberculosis of the penis acquired during circumcision usually due to the ritual of sucking the bleeding circumcision site. In recent years this practice has been modified by the substitution of an aspirating bulb thus avoiding direct contact.

**Tuberculous Epididymitis** represents the only obvious lesion in the male. If neglected or unattended the process breaks through the tunica albuginea of the testis and involves the latter organ. Tuberculous vesiculitis and prostatitis although found at postmortem are seldom diagnosed in the child on clinical examination. The infection is usually hematogenous in origin may be secondary to



boys while Beer and Hyman (1930) noted only 149 female victims in a collected group of 3836 cases. The facility with which calculi may pass through the short urethra of the female may account for this disparity in relative incidence.

**Etiology**—Numerous causative factors have been advanced in an attempt to explain the as yet unsolved problem of urolithiasis. We shall briefly consider the important theories.

**Diet**—Marked reduction in the incidence of urinary calculi in children has been attributed to the widespread improvement in hygienic and dietary management of children the world over. We have already shown the great disparity in incidence statistics of the present and past century. However there are areas where poverty and famine still exist and where a few staple articles of food are used to the exclusion of all others. In China rice is the main article of diet while wheat maize and millet in India and bread in Mesopotamia are staples. It is thus easily understandable why such stone formation is called the poor man's disease. In our own country Holmes and Coplan (1930) demonstrated the frequency of ease with which urinary concretions are formed following the ingestion of large amounts of citrus fruit juices in Florida and southern California.

**Vitamins**—The role of the vitamins in stone formation has been emphasized by recent studies. Vitamin D deficiency has resulted in the production of calculi in as high as 80 per cent of experimental animals. The calculi are most often phosphatic in composition and Higgins (1933) has demonstrated the production and solution of such stones by utilizing a high vitamin A acid ash diet. Kilham and Grewal (1933a) noted keratinization and desquamation of the urinary tract epithelium with subsequent concretion formation.

Vitamin D in the form of irradiated ergosterol in excessive amounts was used by Dixon and Hoyle (1928) to produce calcium phosphate calculi in animals. These workers noted elevation in calcium and phosphorus excretion and reasoned that the excessive exposure to ultra violet rays of the sun together with a deficiency of vitamin A caused the high incidence of calculi in the tropics.

Calcium rich diets have resulted in calculus formation according to McCollum, Summons and Becker (1929) who felt that this factor was more important than vitamin A deficiency. This observation takes on added significance in view of the frequency with which urolithiasis is found in cases of parathyroid adenoma where high blood calcium is a characteristic finding.

**Infection**—Focal infection has been stressed by Rosenow and Meisser (1921) who demonstrated an apparent specificity of streptococci in the formation of calculi. The organisms isolated from the urines of patients with urolithiasis were inoculated into the tooth pulps of 6 dogs with resultant calculus formation and isolation of

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primary tract tuberculosis and is best treated conservatively. Occasionally, epididymectomy may be necessary.

**Tuberculosis of the Female Genitalia** is even more rarely encountered than the similar condition in male children. Involvement of the uterus, ovaries or Fallopian tubes is secondary to intestinal or peritoneal tuberculosis and their presence is made known either at the time of laparotomy or on postmortem examination. Bruning (1902) reported on 44 collected cases in children and Grafe (1914) added 19 more. Involvement of the external genitals—labia, vagina or clitoris are least often noted and then only in association with similar involvement of the internal genital organs.

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the same organisms from the animal's urine. While Rosenow's theory is not universally accepted foci of infection in the teeth, oropharynx, gastrointestinal tract or genitalia may prove to be etiologic factors and should be removed.

Local urinary tract infections frequently account for initial or recurrent calculi. Brown and Earlam (1933) conclude that 18 per cent of bacilli infecting the urinary tract possess the power of splitting urea and therefore favor the formation in turn of ammonium carbonate and magnesium phosphate. While the bacterium *proteus vulgaris* is the chief urea splitting organism, the same workers showed that 40 per cent of *Staphylococcus albus* strains also possess such power.

Calculus formation occurs more frequently than can be ascribed to coincidence in cases with chronic infection elsewhere in the body of bed ridden children. Long standing immobilization necessitated in bone tuberculosis or pyogenic osteomyelitis is conducive to stone formation due to a combination of infection, stasis and hypercalcemia as a result of decalcification of bone.

**Stasis.** Faulty drainage may result from prolonged immobilization or by existence of actual organic obstruction. Congenital anomalies account for many of these obstructive uropathies. With stasis, infection sooner or later supervenes and the groundwork is laid for calculus formation. This leads to another interesting phase of stone formation.

**Colloid-Crystalloid Imbalance.** Urine is a supersaturated solution by virtue of its normal crystalline content. The colloids protectively prevent precipitation of the crystalline elements by the adsorption of insoluble crystalloids on their surfaces. Infection and the epithelial desquamation produced by vitamin A deficiency or the presence of foreign bodies upsets this fine colloid-crystalloid balance. In addition, excessive crystalloid output following particular dietary excesses or skeletal decalcification may further promote such disturbances with resultant calculus formation.

**Hyperparathyroidism.** While rarely existent in children, this condition is mentioned because of numerous reports on its relation to urinary lithiasis. Albright, Baird and Bloomberg (1934) and others have reported on the relative frequency of adenomata of the parathyroid in association with hypercalcemia, hypercalciuria and urinary lithiasis.

**Types of Urinary Calculi.**—The chief urinary salts which contribute to the structure of stones are uric acid and urates, oxalates, phosphates, and rarely cystine and xanthine. The ammonium, sodium, potassium or magnesium radicals are combined with them in a variety of compounds.

**Uric Acid Calculi.**—Uric acid calculi constitute the majority of vesical calculi in children. Large amounts of uric acid are excreted

during the first two weeks of life. A high purin diet increases uraturia. Precipitation of the crystals in a highly acid urine results in uric acid infarcts with subsequent colic and occasionally hematuria. Uric acid and its monosodium salt may precipitate out of solution in the form of a brick red sediment often mistaken for blood while their abundance may result in the formation of large calculi. The stones are usually hard with smooth or bosselated surfaces showing varying degrees of pigmentation most often yellow.

**Oxalic Acid**—Oxalic acid and its salts frequently give rise to calcium oxalate stones or combine with phosphatic or uratic crystals to form laminated calculi. About 30 per cent of all calculi are partially composed of oxalates chiefly the octahedral calcium oxalate crystals. Oxalates are derived chiefly from exogenous sources a minute endogenous source being from body connective tissue. Intestinal fermentation causes an increased production of the acid and a high purin diet is also a contributory factor in oxaluria. The association of oxalates and urates in the mulberry type calculus is one of some significance in this respect. Other types of oxalate calculi may be smooth pea sized or jack stone in contour. They are characteristically hard and vary in color being brown gray or black.

**Phosphatic Salts**—Abundance of phosphatic salts in the urine produces phosphaturia with a diffuse milkiness that clears on the addition of acetic acid. An alkaline urine is a characteristic finding associated with the thick white sediment which is often made up of rosettes and prisms of calcium phosphate or the coffin lid crystals of magnesium ammonium phosphate.

Phosphatic concretions occur either in conjunction with infections in the urinary tract or with a persistent highly alkaline urine. Overindulgent mothers in forcing fruit juices excessively upon children may promote such a condition since the source of phosphates is chiefly exogenous. Vegetables fruits and potatoes are especially rich in phosphates.

Phosphatic calculi are usually mixed in composition have rough grayish surfaces and are rather friable. Their contour varies with the location of the stone. The stag horn type results from molding of the fused mass to the contour of the renal pelvis.

**Cystin Lithiasis**—Cystin lithiasis represents a metabolic disturbance in the failure of the body to break down this essential amino acid with its subsequent unaltered excretion in the urine. Wollaston (1810) first noted the condition in a child of five and a man of thirty six years following analysis of calculi removed from their bladders.

Cystinuria which promotes calculus formation is usually congenital and often hereditary and familial. Children may consistently show the small flat hexagonal cystin crystals in an acid urine without any symptoms. However obstruction stasis and infection

provide the groundwork for such calculus formation in the child Seeger and Kearns (1925) collected 181 cases of cystinuria with 125 having calculi Fortunately the condition is rare

**Xanthin Lithiasis** — Xanthin lithiasis is a decided rarity Polkey (1934) cites 17 cases of pure xanthin calculi the element is also found in association with the more common stone-forming crystals Xanthin representing an arrested stage in uric acid metabolism is found in coffee tea cola nut cocoa and the diuretics caffeine theobromine and theophylline

**Pathology** — Whether the etiology of the stone be due to faulty metabolism disturbed crystal equilibrium stasis or infection the underlying pathologic changes are the same Mechanical irritation ulceration of the mucosa and infection usually follow although the same findings associated with antecedent infection or tumor may be the cause rather than the effect of calculus formation Obstruction and pressure atrophy may follow upon progressive enlargement of calculi especially in cases of renal and vesical stones In children anomalies obstructions tumors and foreign bodies all of which favor stone formation are common associated lesions

**Renal Calculi** — Renal calculi are uncommon in children In Wile's series (1930) of 864 renal calculus patients 14 were children and Bokay and Brown (1927) found 9 in 1836 collected calculus cases Since *ureteral calculi* are invariably of renal origin their incidence is correspondingly less Moreover the ureter in children is comparatively larger and more elastic thus favoring the uneventful passage of calculi into the bladder

**Vesical Calculi** — Vesical calculi account for the major portion of urinary tract calculi in children Bokay and Brown found 1819 such cases in a series of 1836 children with urolithiasis

**Urethral Calculi** — Urethral calculi are rare and occur practically always in males the short female urethra being no barrier to their passage This also explains the lower incidence of vesical calculi in female children

**Symptomatology** Renal calculi may give no symptoms until advanced destruction of the renal parenchyma has occurred Dull pain or colic often mistakenly considered intestinal in origin may occur in older children Radiation of the pain to the groin or external genitalia should make one suspicious of renal or ureteral stone Urinary difficulties may manifest themselves due to secondary vesical or urethral involvement Anuria may be the first sign of a previously inactive calculus Pyuria is a frequent finding while hematuria is relatively infrequent One may find brick red particles of uric acid in the urine and gross fragments or microscopic crystals of different composition

Vesical calculi are characterized by signs of bladder irritability

with associated dysuria frequency, urgency interruption of the stream or enuresis. Pyuria is usually noted while hematuria occurs less often. Reflex priapism may occur due to posterior urethral irritation.

Urethral calculi are almost always accompanied by urinary difficulties. In lower tract calculi the role of foreign bodies as an etiologic factor should not be overlooked. In all cases the systemic reaction due to urinary tract infection may mask the underlying calculus disease. Chills fever malaise failure to gain in weight anorexia and reflex gastric irritability may be prominent symptoms of urolithiasis.

**Diagnosis**—An orderly investigation will invariably lead to accurate diagnosis localization and choice of treatment of urinary tract calculi. Such a study includes consideration of

1 History—with emphasis on the presence of lumbar pain colic digestive symptoms difficulties in urination gross pyuria or hematuria.

2 Physical examination—including review of the systems careful bimanual palpation of the abdomen and loin inspection of the external genitalia and rectal examination.

3 Urinalysis—including microscopic study for red and white cells and crystals.

4 Plain roentgen ray—Thomas and Tanner (1922) reported 112 calculi of a collected series of 203 discovered by this means.

5 Cystoscopy—with actual visualization of urethral or vesical calculi.

6 Kidney function tests.

7 Ureteral catheterization and retrograde pyelography.

We favor the retrograde injection of contrast media with antero posterior and body shift oblique radiographic exposures over intravenous urography. This procedure not only removes any doubt as to definite localization but is of value as a guide to therapy. Associated lesions if present are revealed and the extent of the destructive process associated with the calculus ascertained.

**Differential Diagnosis**—This is chiefly concerned with excluding appendicitis and intestinal colic associated with gastro intestinal upsets. In children the classical signs and symptoms of appendiceal irritation may be absent. In such instances careful urinalysis to exclude the presence of undue numbers of leukocytes either discrete or clumped and red blood cells may be the deciding factor. An appendix overlying the ureter as it courses over the pelvic brim may cause pain simulating typical ureteral colic. When in doubt, laparotomy should be done despite the fact that a normal appendix may be ultimately found. Intestinal colic is usually accompanied by a history of similar occurrences or of definite dietary indiscre-



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tion The pain is generally shorter in duration does not radiate in the characteristic manner of calculus and is frequently accompanied by diarrhea

**Treatment of Urolithiasis**—The conservative management of childhood urolithiasis is essential A medical regime which includes forcing of fluids urinary antiseptics and dietary regulation should precede any operative intervention and should also be used post operatively as prophylaxis against recurrence A large calculus may offer no alternative treatment other than its surgical removal

Persistent phosphaturia or the finding of phosphatic stones calls for limitation of foods with high phosphate content restriction of calcium rich foods and acidification of the urine To this end an acid ash diet may be prescribed with limitation of green vegetables fruits potatoes egg yolk milk and lentils

In uraturia and oxaluria or the actual incidence of calculi restrictions are placed on purin rich foods (sweetbreads gelatin liver and bouillon) and those with high oxalate content (cocoa chocolate spinach rhubarb red beets and potatoes) An alkaline urine is desirable and may be attained either by suitable medication an alkaline ash diet or both combined

The rare cystin calculus victim is helped by alkalinization of the urine Dietary limitation is difficult since almost all proteins contain cystin In this connection it is well to emphasize the necessity for maintaining an adequate well balanced diet in children Better results are obtained by such a policy than by enforcing the far from proven rigid restrictive diets

Diagnostic instrumentation may be modified or extended to include therapeutic steps Cystoscopy combined with use of foreign body forceps dilating ureteral instruments or use of the lithotrite may serve the operator well and spare the child in operation The final therapeutic step involves surgery adapted to the part of the tract involved

Renal calculi may be so small as to merit only observation conservative medical measures or repeated pelvic lavages in the hope that dissolution or passage of the calculi will follow In children uretero pelvic anomalies due to aberrant vessels and the like may necessitate open operation for removal of the calculi as well as the predisposing lesion

**Operative Procedures** The nature of the operation depends upon individual findings Pyelotomy nephrotomy nephrostomy resection of the kidney or nephrectomy may be indicated to remove not only the stone or stones but also the associated lesion be it congenital hydronephrosis pyonephrosis or tumor

Sound judgment is all important in the treatment of juvenile urolithiasis Bilateral renal involvement and multiple unilateral

calculi often taxes the surgeon's acumen. Treatment cannot conform to any general rule. We agree with Human (1935) that a frequency of over 30 per cent of nephrectomy in all reported series of cases is too high. With our meager knowledge of the exact etiology of stone formation leaving a child with one kidney is worthy of considerable thought and conservatism. The removal of associated and possible contributing lesions in the urinary tract is essential to the prevention of recurrence of calculi.

*Ureteral calculi* are uncommon due to the dilatibility of the structure in children. A congenitally small ureteral orifice or stricture of the intramural portion of the ureter may provide an impassable barrier for a stone. Beer feels that so called congenital megalo-ureter in some instances is due to prolonged impaction of a stone in this segment.

Progressive ureteral dilatation with instillation of a lubricating solution to facilitate passage of the calculus may be successful. Merotomy of the constricted ureteral orifice may open the way to passage of the calculus or may bring the stone into view and permit grasping it with foreign body forceps. Multiple ureteral catheters and special extractors find little application in children. Ureterolithotomy is seldom necessary unless a large calculus is so impacted or lodged in a diverticular pouch as to make treatment by more conservative measures ineffective.

*Vesical calculi* are best treated with full knowledge of etiologic possibilities. Correction of dietary deficiencies may lead to rapid dissolution of the calculus. Lavage of the bladder with a solution of such hydrogen ion concentration as to promote disintegration of the aggregated crystals may favor dissolution. A foreign body acting as a nidus for crystalline deposition demands special consideration as does any organic obstruction at the vesical neck or urethra be it acquired or congenital. Diverticula, adynamic or dynamic sphincteric disturbances, posterior urethral valves or strictures and kindred lesions have accounted for a number of cases of vesical calculi and should be eradicated surgically prior to or simultaneous with the removal of the bladder stone.

Removal of vesical calculi may sometimes be effected through the cystoscope by the use of foreign body forceps or by litholapaxy. Joly (1929) outlining the limitations of use of the lithotrite in children states that stones of 1 to 1.5 cm in diameter may be safely crushed with the No. 10 F or 12 F instrument and those 3 to 4 cm in diameter by the No. 14 F to 18 F size. Litholapaxy requires specialistic skill and when in doubt suprapubic cystostomy should be the procedure of choice.

Suprapubic cystostomy is always indicated in children when conservative measures have failed. The operation of cystolithotomy

is a simple one and while the bladder may be closed immediately and an urethral catheter left indwelling it is always advisable in the presence of cystitis to drain the organ for a short time as a therapeutic measure in clearing up the residual infection. Once the supra pubic tube is removed and providing a free urethral channel exists, closure of the wound occurs with remarkable rapidity.

*Urethral calculi* may be amenable to local manipulation using endoscopic forceps or lubricating solutions. A posterior urethral calculus may by pushing it backward be converted into a vesical calculus. Impaction of a stone is usually caused by stricture, valve or diverticulum formation and correction of such conditions usually result in easy passage or removal of the stone. Internal or external urethrotomy may be ultimately necessary in the case of a persistently impacted urethral calculus.

## CHAPTER XLI

### THE KIDNEYS

#### ANOMALIES OF THE KIDNEYS

MAI FORMATIONS of the kidney represent one of the most frequent congenital defects. The variety of such occurrence is legion although often of purely academic interest. In certain anomalous developments, the sequelae are manifested clinically and then assume major proportion necessitating surgery.

The rôle of anomalies in disease production is evident in the findings of Campbell (1936), who found 179 such instances in 580 cases of chronic pyuria in children—an incidence of 30 per cent. Statistical studies have consistently shown the markedly increased ratio of urinary tract disease as compared to the routine postmortem incidence.

Maldevelopments of the kidney and ureter predispose to pathology. Obstruction, infection and stone formation are not uncommon. Although the kidney originates from ureteral as well as nephrogenic sources, maldevelopment of one structure will affect the other.

The subject of urogenital anomalies has already received general consideration and we shall therefore concern ourselves with specific maldevelopments of the kidney.

**Variations in Number.**—Total absence of renal substance is, of course, incompatible with life and is found only in monstrosities at birth.

**Solitary Kidney**—Solitary kidney assumes clinical and surgical importance by virtue of the fact that prior to modern urologic diagnostic refinements, and even today when such means are not employed, the sole remaining kidney has been unwittingly sacrificed with subsequent anuria and death.

Agenesis of the kidney occurs once in every 1000 to 4000 autopsies. Collins (1932) collected 572 cases from the literature, reporting 9 of his own. Males outnumber females and left-sided absence is more common than right-sided agenesis. In the majority of cases the ureter and its orifice are not demonstrable but the adrenal on the affected side is usually present.

Associated genital, anal or other bodily defects are not uncommon. The handicap of harboring a solitary kidney manifests itself in pathologic involvement of the organ by infection, stone or injury. Nephrotomy, hemisection or pnelotomy has been performed on such diseased kidneys with uneventful recovery.

**Multiple or Supernumerary Kidneys** are rare. Kretschmer (1929) collected 30 cases of a third kidney, 22 in life and 8 at postmortem examination. The condition is not to be confused with the more common double kidney, the renal parenchyma of which is not completely separated from its unilateral mate.

**Double Kidneys** Double kidneys develop as a result of anomalous changes at the cranial or caudal end of the ureteral bud. The ureter of the accessory kidney may enter the twin structure of that side or make its own entrance into the bladder or even have an extra vesical outlet.

Double kidney, as above differentiated from the distinct supernumerary organ, frequently manifests itself by duplication of pelvis and ureter. Interference with drainage and greater susceptibility to infection often makes the condition a problem both as to accurate diagnosis and treatment. Numerous instances of successful heminephrectomy or ureterectomy have been recorded for pathology in such fused structures.

**Variations in Size — Hypoplasia** — Hypoplasia of the kidney assumes importance in the presence of malfunction of its mate. Varying degrees of hypoplasia may occur from a tissue rest hardly demonstrable grossly to a small contracted organ that necessitates differentiation from acquired atrophy. The ureter may be absent or represented by a fibrous cord. Among other etiologic factors, anemia of the organ due to defective vascular supply has been cited.

The opposite kidney usually shows a development beyond its normal size. This is attributed to embryologic substitution rather than to compensatory hypertrophy. Diagnosis of unilateral hypoplasia may be difficult since sufficient functioning tissue frequently remains to give fair qualitative function tests. Quantitative tests combined with plain and pyelographic roentgen ray studies may demonstrate impaired total function and a small renal pelvis surmounted by a rim of renal tissue.

Treatment varies with the organ involved. If the hypoplastic organ be involved, nephrectomy will be the operation of choice since the reserve powers of the rudimentary organ are negligible. On the other hand, the opposite kidney, in the presence of disease, should be considered as a solitary kidney and treated with extreme conservatism.

**Hyperplasia** Hyperplasia of the kidney, with actual increase in renal constituents as opposed to compensatory hypertrophy, may occur. This is true in the case of very early dysfunction (*in utero*) of the renal mate. The issue, however, is purely academic and entails no clinical significance.

**Variations in Form — Asymmetry** — Asymmetry of the kidney, as represented in a long, short, broad or lobulated structure, is merely of anatomic interest.

*Fusion* of the kidney may result in a variety of forms

- 1 L-shaped with one organ at right angles to the other
- 2 Sigmoid with the upper pole of the transposed kidney joined to the lower pole of its mate (Incidence 1 to 8000)
- 3 Disc or pancake type with partial to complete fusion and median location of the fused structure
- 4 Horseshoe kidney

**Horseshoe Kidney**—The horseshoe kidney represents the commonest type of fusion. It occurs once in every 500 to 800 autopsy cases but with a greater clinical incidence since such organs are more vulnerable to disease processes. Anatomically and pathologically the horseshoe kidney varies. The union may occur above with the concavity below or more commonly below with the concavity above. The joined kidneys then take on an L shaped configuration. The joining bar or isthmus of tissue crossing the vertebral column may be fibrous or may contain renal parenchyma. The number of pelvis and ureters may vary from one to as many as four.

*Diagnosis*—While made preoperatively in many cases the condition is also frequently missed and discovered only at operation. Clinically pain in the renal or umbilical area, gastro-intestinal disturbances or urinary difficulties may be manifested. Physical examination may reveal a palpable mass or even the isthmus in thin relaxed individuals. A flat roentgen ray plate and ureteropyelograms will facilitate diagnosis by depicting

- 1 Poorly defined upper or lower poles due to fusion
- 2 Partial obliteration of the psoas muscle shadow by the isthmus
- 3 Variation in pelvic contour and direction of calices which are often directed toward the spine

*Treatment*—This only assumes importance when obstruction, pyogenic infection, tuberculosis, tumor or calculus occurs. These complications are usually missed by children but occur in adults in an incidence greater than that seen in normal organs.

**Variations in Position** **Faulty Rotation**—Faulty rotation of the kidney, either incomplete or excessive in type, may produce significant obstruction. Pyelography may demonstrate the pelvis and ureter anteriorly in incomplete rotation or descending behind or lateral to the kidney in cases of reverse rotation. Frequent associated lesions include aberrant vessels, ectopia or renal fusion.

If pyelography shows dilatation of the renal pelvis (pyelectasis) with blunting of the calices, surgery is indicated as a conservative measure. It may necessitate severing aberrant obstructing vessels, plastic repair of the pelvis, nephropexy or in extreme cases nephrectomy.

**Ectopia of the Kidney**—Ectopia of the kidney represents a congenitally displaced organ which has never occupied its normal position. This distinguishes it from the movable or floating organ.



most frequently seen in adults. Thomas and Barton (1936) from a survey of the literature noted an incidence of 1 in 822 autopsies and 1 in 547 urologic examinations.

The ectopia may be

1 Unilateral

2 Bilateral

3 Crossed. This involves the displacement of one kidney to a position below the opposite one. Fusion is a frequent finding in these cases giving rise to the sigmoid kidney. Such displacements predispose to obstruction, infection or lithiasis.

The location of the ectopic kidney varies from upward displacement into the thorax associated with congenital diaphragmatic defects to lumbar, iliac, pelvic or median positions.

**Symptomatology.** The condition may be symptomless or associated with superimposed pathology in the displaced organ. A mass may be present in the pelvis and give rise to pressure symptoms. Bimanual abdominal, vaginal or rectal palpation may assist in correct diagnosis. Conclusive evidence is furnished by cystoscopy, ureteral catheterization and pyelography. Careful differentiation in the case of pelvic tumors is warranted since more than once a perfectly normal pelvic kidney has been removed during the course of exploratory laparotomy where urologic investigation had been omitted.

**Treatment.** This depends on the nature of the disease process and the location of the organ. Transperitoneal approach may be most adaptable in dealing with ectopic kidneys in the pelvis.

**Variation in Vascular Supply.**—The arterial supply of the kidney varies considerably as to number and point of origin. Normally the renal artery is single subdividing into three to five branches at the hilum. Abnormally upper and lower pole vessels may occur with their point of origin being from either the aorta, renal artery, external iliac, hypogastric, middle sacral, lumbar, spermatic, pancreatic, right colic, hepatic or inferior phrenic arteries. There may be two or more arteries to the kidney from any of the above mentioned sources.

Associated venous anomalies paralleling the arteries are frequent. They may arise from the upper or lower pole of the kidney and empty into the renal vein proper or into the vena cava or iliac vein. Retropericolic veins occur with disturbing frequency at times.

Such vascular anomalies are a source of trouble both as a factor in producing obstructive uropathies and also as a serious complication during surgical procedures. Huge hydronephrotic kidneys may eventuate from insidious obstruction produced by an aberrant vessel crossing at the uretero-pelvic juncture. (fig. 233.)

Severing of the constricting vessels or plastic side tracking of the urinary stream where it is deemed inadvisable to cause any possible

circulatory embarrassment is indicated. Profuse hemorrhage may occur during the course of surgery on the kidney by accidental severance of an anomalously placed vessel. Just as the vessel's presence was unheralded so is its elusiveness in attempting hemostasis. The result is often catastrophic. Packing may be a life-saving measure or a clamp may be left *in situ* and the wound closed about it where an attempt at ligation and renal removal is deemed too great a risk. The hemostat may be removed in forty-eight to seventy-two hours or allowed to remain until the clamped tissue sloughs off.

**Variations in the Renal Pelvis** — These are of no significance other than morphologic. Normal structural variations are numerous. The presence of an intra- or extrarenal pelvis as delineated by pyelography is of assistance to the surgeon in his choice of operative approach. Thus if a large calculus is present in a kidney with a small intrarenal pelvis nephrotomy would be preferred to pyelotomy. Pyelectasis caused by obstructing vascular cords has already been cited. Diverticulum of the pelvis has been reported but most of these are deformities due to associated pathology rather than true pouches.



FIG. 233. Advanced hydronephrosis due to uretero-pelvic contraction by an aberrant vessel in a boy age eight years. Nephrectomy was performed. (Courtesy of Dr. S. R. Woodruff.)

**Congenital Hydronephrosis** — This condition merits consideration as a secondary manifestation of interference with the outflow of urine from the renal pelvis. It may be due to abnormal insertion of the ureter into the pelvis, to aberrant vessels constricting the ureter usually at the uretero-pelvic juncture, to idiopathic constriction of the upper ureter, or to anomalous ureteral valves. Production of the typical sack kidney begins *in utero* or in early infancy and often manifests itself symptomatically before the age of five years. Its progress is unimpeded until its size or superimposed infection attracts attention.

**Symptomatology** The hydronephrotic sac may assume such size as to produce a pot belly deformity. Pain due to pressure is often a prominent symptom in addition to abdominal asymmetry. Gastro-intestinal complaints are common and often mask the underlying pathology. The urine may be clear or laden with pus depending upon the presence or absence of infection. The onset of infection is characterized by fever, pain and urinary symptoms.

**Diagnosis** This may be made at the time of cystoscopy and ureteral catheterization. An *impasse* may be encountered along the course of the upper ureter or at a point corresponding to the uretero-pelvic juncture. Retrograde pyelography will further confirm such an observation and will also supply additional information as to the size and extent of the hydronephrotic sac by means of contrast radiopaque media. Differentiation from cystic disease and tumor is usually not difficult. Rupture of these thin walled sacs is more than a theoretical possibility. Direct or indirect trauma has resulted in such complications and Herman (1921) collected over 20 cases from the literature and added 1 of his own. Similar instances find their way into the medical journals sporadically.

**Treatment**—This depends on the nature of the obstruction and the extent of renal parenchymal destruction. In all cases the opposite kidney should be studied for function and to exclude similar pathology since the process is occasionally bilateral. Conservative operative repair with pyeloplasty or reimplantation of the ureter may suffice. Nephrectomy on the other hand may be the only procedure applicable to advanced lesions.

**Prognosis** is good if the condition is unilateral and infection is absent or minimal. Many of the children die within the first six months of life and huge unilateral or bilateral congenital hydronephrosis is found at autopsy.

### INJURIES OF THE KIDNEY

Despite the protected position of the kidney within the bony and fleshy framework of the trunk, traumatism to it is not uncommon. The increase in vehicular traffic has elevated the incidence of accidents which directly or indirectly cause such lesions. Direct blows, falls or kicks to the lumbar or anterior abdominal regions may result in severe renal injury. Such accidents while riding go-carts, bicycles, sleds or playing strenuous games involving body contact are possible sources of injury to the kidney.

Apart from isolated trauma to the kidney, one may find fractured ribs, injured pleura, diaphragm, liver, spleen, intestines or bladder. A factor which enhances the effect of trauma in children is the presence of a congenital abnormality such as hydronephrosis, secondary to an aberrant blood vessel, polycystic disease or ectopic

kidney Lazarus (1931) operated upon boys of six and sixteen years of age for spontaneous rupture of hydronephrotic organs

**Pathology**—The pathology of renal traumatism varies with the nature of the blow and the relative vulnerability of the kidney by virtue of its location and normal or abnormal state. The possibilities and their sequelæ are herewith listed

1 Tear of the fatty capsule which may pass unnoticed

2 Contusion with or without rupture. These are serious only when the kidney is the seat of preëxisting pathology or infection

3 Parenchymal injuries involving the capsule. These may be characterized by severe hemorrhage due to pulping of the kidney

4 Pelvic injuries. When associated with parenchymal tears such rents are extremely dangerous due to hemorrhage and urinary extravasation. Lacerations of the vascular pedicle, ureter or even total avulsion of the kidney has occurred with rapid exitus

5 Peritoneal tears associated with pelvic injury are fortunately rare but find their highest incidence in children under ten years of age

6 Stab or gunshot wounds although usually associated with warfare are occasionally seen following playful or willful use of such instruments by children

**Symptomatology**—A history of the nature of the injury may be of some assistance. Physical examination may reveal superficial ecchymosis or a palpable tender loin mass (hematoma) or extreme rigidity and exquisite tenderness localized to the renal area or diffuse over the whole abdomen

The extravasation of blood and urine may gravitate along retroperitoneal tissue planes and produce swelling and ecchymosis of the external genitalia. The salient features of the picture in children are shock, local pain, tumefaction and hematuria complicating signs of peritonitis, ruptured abdominal viscera or hemothorax may be associated

The symptoms vary with the extent of the injury. Hematuria is present in almost all cases, rare exceptions being where the ureter is completely severed or plugged by clots. The degree of shock is exaggerated in children as compared with adults. Anæmia or vomiting, tympanites or paralytic ileus may occur reflexly. A rare complication is reflex anuria

**Diagnosis**—This is made from the history, physical examination, plain and intravenous urography and in selected cases cystoscopy and meatoscopy. The status of the opposite kidney should always be ascertained. Gambling on its condition when opportunity for investigation is available is assuredly unwarranted. All findings must be coordinated since one or more separately may fail to be informative. Occasionally intravenous urography will fail to show excretion of the injected dye from the affected side due to reflex inhibition of the secretory-excretory mechanism

1 Urogenous infections in a kidney previously affected by anomaly obstruction hydronephrosis tuberculosis neoplasm or lithiasis

2 Metastatic cortical coccal infections with primary foci elsewhere in the body and secondary involvement of a previously healthy kidney

**The Urogenous Group**—The condition usually manifesting itself in pyonephrosis engrafted on any one of the enumerated disease processes needs little consideration. Each of the urologic precursors of the infection is described elsewhere as are the common obstructive abnormalities. In children obstructive uropathies with superimposed infection are the most common conditions requiring surgery.



FIG. 234.—Intra-encous urogram showing bilateral reduplication of renal pelvises. This 4½-year-old girl was investigated for persistent pyuria.

**Treatment** This is concerned with determination of the nature and extent of the infection; conservative measures including cystoscopy, ureteral catheterization, pelvic lavage and instillation of medication and finally other methods proving unavailing surgery. Incision and drainage, renal decapsulation, nephrostomy or nephrectomy may be resorted to in an attempt to promote healing or to completely eradicate the infection.

Campbell (1933) dealt with 6 cases of hemipyonephrosis in children with anomalous renal duplication or so called double kidney. Four of these patients were less than thirty months of age; 1 three



FIG. 245 Left retrograde pyelogram showing clear delineation of ureter and pelvis



FIG. 246 Right retrograde pyelogram showing lower uretero-pelvic constriction  
Colon bacilli were found in abundance in the urine specimen from this side  
(684)

and another five years old Nephrectomy was performed on 3 while the remainder were subjected to uretero heminephrectomy. Such case reports add to repeated observations that kidneys with congenital defects are much more susceptible to infection than are normal ones (Figs 234 235 and 236)

**Metastatic Cortical Coccal Infections**—These may pursue any one of three courses. The infection may occur as a diffuse nephritis mild or severe in character. The mild type runs an uneventful course while the acute fulminating type may necessitate nephrectomy promptly. Such an organ is found to be diffusely infiltrated edematous and extremely friable. In milder types the kidney may appear normal superficially but decapsulation will reveal points of adherence and small areas of suppuration.

**Multiple Cortical Abscesses** represent a more advanced stage of the focal nephritis described. The suppurative focus may be limited to one portion of the kidney. Superficial rupture of cortical abscesses are usually precursors to perinephric suppuration.

**Carbuncle of the Kidney** typifies the end stage of coccal infections. Multiple abscesses may coalesce and form a circumscribed tumefaction. Perinephric abscess described elsewhere arises from such a source.

**Etiology**—The staphylococcus is the offending agent in most cases and the focus of infection may be the skin (cellulitis furuncle or carbuncle) paronychia or felon osteomyelitis or tooth abscess. An antecedent history of such lesions especially in the child is difficult to elicit. Finding a superficial scar of a pyogenic infection which has come and gone may be the only clue available.

Aschner (1936) reviewed 61 cases in all age groups with 8 under thirteen years of age. Beer (1936) added 43 cases in whom operation was performed. Males were afflicted three times as often as females in Aschner's series. The disease was usually unilateral without particular predilection for either kidney.

**Symptomatology**—The onset may be mild or acute. Fever and dull to sharp pain in the flank or abdomen may characterize the condition which has been mistaken for influenza pneumonia typhoid tuberculosis intra abdominal pathology Pott's disease of the spine or hip joint involvement. Linder (1929) and Beer (1936) have stressed the point that general surgeons see more of these cases than urologists because of the frequency of abdominal signs. Suppuration with extension onto the anterior aspect of the kidney may give signs of peritoneal irritation and abdominal muscle rigidity. Lower pole involvement often simulates appendiceal or pelvic pathology while upper pole lesions resemble subphrenic abscess or diaphragmatic pleurisy.

Irritability malaise loss of weight pallor nausea or vomiting low grade fever or a septic appearance may be noted in the child.

History of antecedent skin suppuration is helpful. Physical examination may reveal a loin mass, muscle rigidity, diffuse or point tenderness in the renal area or costovertebral angle. A positive Murphy percussion test may be elicited. Cystoscopy, urinalysis and roentgen ray studies may be of no assistance. A carbuncle may distort the pelvic contour in pyelography and the inflammatory process may obliterate the clearly defined psoas muscle margin together with curvature of the spine away from the affected kidney. Difficulty in diagnosis is evidenced by the observation of Lazarus (1919) that only 4 of 22 collected cases of carbuncle of the kidney were diagnosed preoperatively.

**Treatment.** Local coccal infection should be treated conservatively since the majority run their course with complete healing. However, this may prove deceptive in that several weeks later the same patient may show signs of localized renal suppuration. Fulminating nephritis represents a grave condition and demands early nephrectomy. If a staphylococcus infection of the kidney is suspected and the patient is not responding to conservative measures exploration of the organ is indicated. Exposure of the kidney is not sufficient since small foci may underlie the capsule. Hence decapsulation is advisable to permit free drainage.

If multiple or even localized areas of suppuration are exposed incision and drainage after decapsulation is sufficient. In certain instances resection of a portion of the kidney may be beneficial. Placing rubber dam anterior and posterior to the decapsulated kidney and avoiding tight closure of the loin wound will facilitate free drainage. Nephrectomy should be avoided or done as a secondary procedure since one is never certain if the same process does not already involve the opposite kidney.



**Echinococcus or Hydatid Disease**—This is caused by the parasitic tapeworm *liver echinococcus*. While the kidney is the favored site in urogenital infections of this type the organ is involved in only about 3 per cent of all cases of the disease. Furthermore the fact that it takes from fifteen to twenty years for cysts to manifest symptoms makes the condition an adult rather than a pediatric urologic problem.

**Actinomycosis**—Involvement of the urinary tract by actinomycetes or ray fungus is rare but worthy of mention because of the need for diagnosing obscure cases which prove baffling until the etiologic agent is found. Cumming and Nelson (1929) collected 37 cases of renal actinomycosis from the literature. This number represents all age groups. Kretschmer and Hibbs (1936) after scrutinizing the literature found 4 cases of renal actinomycosis in children adding 1 of their own.

The usual portal of entry for the ray fungus is the buccal cavity following local trauma. The head and neck are especially common sites for the infection which extends along fascial planes and rarely via the blood stream or lymphatics. Involvement of the kidney alone is termed a primary infection in the absence of any other demonstrable focus although it is probable that all urogenital infections are fundamentally secondary ones. The testicle and seminal vesicles (Hinman 1935) have been reported as sites of genital involvement.

**Pathology**—Actinomycosis produces a chronic suppurative process characterized by the presence of light yellow or orange sulphur granules. The granules comprise masses of filamentous Gram positive organisms often arranged as club shaped filaments radiating from a nucleus. The kidney is usually involved through extension by continuity from a pulmonary or gastro intestinal lesion.

**Symptomatology**—Symptoms are vague and suspicion should be aroused by the presence of a draining sinus adjacent to the renal area. Fever abdominal pain malaise loss of weight or local tumor formation may be noted. Diagnosis can only be made by demonstrating the fungus. Clinical studies including pyelography may lead to a mistaken diagnosis of tuberculosis or tumor only to be corrected postoperatively or postmortem.

**Treatment**—Nephrectomy is indicated in unilateral uncomplicated lesions. Large doses of potassium iodide orally and 2 per cent copper sulphate solution for irrigation of the sinus tract have proven efficacious. Hunt and Mayo (1931) cured 5 of 7 cases by nephrectomy. Kretschmer and Hibbs case a girl aged ten years was well two years after nephrectomy.

**Lithiasis**—Calculus disease of the kidney represents but one phase of the broader subject of urolithiasis. Its etiology diagnosis and treatment will be found under the general heading of Urinary Lithiasis.

## CYSTS OF THE KIDNEY

Without disregarding the possibility of congenital anomalous origin placing cystic lesions in the category of renal tumefactions is justifiable from a clinical standpoint

**Solitary Serous or Hemorrhagic Cysts**—These are rare in early life. Since they produce few if any symptoms until adult life recognition is usually made only at autopsy. Kretschmer (1920) noted 4 juvenile cases in a series of 48 cases of solitary cysts of the kidney.

**Polycystic Disease**—This congenital anomaly represents an important factor in the differential diagnosis of renal tumors. Hyman (1930) cites 18 cases in new born and infants up to one year of age and 11 in the next two decades of life in a series of 219 cases.

Doubt as to the origin of the condition is evident from the variety of hypotheses dealing with its production. Virchow held developmental errors such as fibrosis and atresia of the papillary ducts accountable. Malunion of ureteral and metanephrogenic units, uriferous tubule persistence, arrested development of the glomerulus and Bowman's capsule, persistence of mesonephric tubules with transformation into cysts and finally actual neoplastic origin have been promulgated as etiologic factors by other individuals. The developmental defect hypothesis is favored by many because of the frequent association with other congenital anomalies and the hereditary nature of the disease.

**Pathology**—The multicystic lesion is found in both kidneys although in an early stage it may be unilateral. The surface of the kidney is characteristically covered with variable sized blebs often translucent and on section clear viscid fluid escapes from the thin walled sacs. The cysts do not communicate with the pelvis and their progressive enlargement produces pressure atrophy of the renal parenchyma.

**Clinical Course**—Infantile polycystic disease may prove fatal at any early age as contrasted with the latent form which manifests itself in adults. Death is usually due to renal insufficiency. Nausea, vomiting, tympanites or convulsions may herald the presence of such lesions. Bilateral renal tumefaction coupled with urinary output of low specific gravity, albumin, casts and microscopic or macroscopic hematuria may be the first signs of polycystic disease. Plain and retrograde urography will confirm the diagnosis by depicting typical alteration in the normal configuration of the renal pelvis due to elongation and distortion of the calyces.

**Treatment** This is merely palliative. Some, considering the disease hereditary, advise victims of the malady against procreation. Nephrectomy for the condition is contraindicated. Occasionally the cysts may assume such proportion as to cause pain or obstruct

tion by extrinsic pressure on the ureter. In these cases puncture of the cysts is efficacious in relieving obstruction pain and even more important in sparing further pressure atrophy of life saving parenchyma.

### TUMORS OF THE KIDNEY

**Benign Tumors**—Solid capsular growths are very uncommon in children. The reported cases include lipoma, myoma, fibroma, lipoma, fibroma, sarcoma and teratoma. The tumors occur early in childhood with a high incidence among females. The symptoms are vague and the differentiation from other renal lesions is difficult even by cystoscopy and roentgenography. The growths may be amenable to surgical enucleation without disturbing the kidney.

**Malignant Tumors**—Sarcoma invades the kidney and adrenal substance. The operative mortality is high and the ultimate prognosis poor. Preliminary as well as postoperative deep roentgen ray therapy is a useful adjuvant.

**Malignant Embryonal Tumors**—This type comprises the most common malignant renal neoplasm. Himm and Kutzman (1924) found 14 cases in 20,470 children or 1 in 2,000. By comparison the same authors noted 128 renal tumors in 47,000 adults an incidence of 1 in 400 or six times as many as in children. Nevins (1926) collected 145 cases of malignant tumors in children. Of interest is the fact that 11 of these were found in fetuses or newborn and approximately 75 per cent were under five years of age.

**Pathology**—Lesions of the renal pelvis most often of the squamous-cell type are extremely rare in children. The histogenesis of parenchymal tumors is a debatable subject. To avoid confusion they may be classified as embryonal, adenomas, sarcomata or mixed tumors of Wilms.

Microscopically epithelial nests, embryonal tubules, smooth or striated muscle fibers, elastic or myxomatous tissue, fat, cartilage and bone may all be visible. Grossly the firm or fluctuant mass may assume huge proportions and while the average weight is under 5 pounds masses as heavy as 36 pounds have been reported. The malignancy invades adjacent structures and metastasizes via the lymphatics and blood stream.

The etiology of malignant renal tumors is just as confused as their pathology. Trauma plays a negligible role in their production. Displaced Wolffian body tissue, aberrant metotome and sclerotome cells and actual origin from embryonic tissue of the true kidney (as opposed to extrarenal inclusions) represent the three main etiologic hypotheses.

**Symptomatology**—Tumefaction in the renal area is the commonest sign. The mother or nurse may accidentally palpate the mass or may note a superficial asymmetry of the abdomen. The

alert pediatrician in his routine physical examination may discover the mass on abdominal palpation. Recently such a finding was made by a fellow pediatrician on routine examination of a three-months old child and prompt preoperative irradiation extirpation of the tumor and postoperative roentgen ray therapy were instituted. The deadliness of the lesion is best attested to by the fact that eight months later the child was dead.

In addition to tumefaction pain due to pressure weakness vomiting and least often hematuria are noted. In contrast hematuria occurs in 60 to 80 per cent of adults with renal malignancy. Generally speaking the outstanding triad of symptoms in children is tumor pain and hematuria whereas in adults the sequence is exactly reversed.

The clinical course in children is rapid. The tumor grows rapidly, and directly or indirectly accounts for signs of toxemia (malaise diarrhea vomiting anemia weight loss and fever) or of pressure (dyspnea ascites, edema of the lower extremities or symptomatic varicocele). Examination of the abdomen may reveal a mass which is small and freely movable or fixed and filling the upper abdomen with bulging flank.

**Diagnosis**—This is usually obvious since the child is brought to the doctor late. History physical examination cystoscopy and urography will contribute to a final diagnosis. (Fig. 237.) If the appalling death rate of renal malignancy is to be reduced earlier diagnosis is essential. Persistent gastro-intestinal symptoms and undetermined intermittent fever may represent premonitory signs while careful abdominal palpation may uncover early renal tumefaction. It must be emphasized that negative urinalysis does not rule out renal tumor. In addition to the routine urologic examination roentgen ray of the chest for possible metastasis should be done.

Differential diagnosis presents several interesting problems. The urologic investigation will usually exclude congenital hydronephrosis pyonephrosis tuberculosis or calculus disease. Combined with careful abdominal palpation and blood counts it will also exclude hepato-splenomegalies. Solitary cysts of the kidney are rare while polycystic disease is usually bilateral. Neuroblastoma and tumors of the renal capsule or retroperitoneal spaces may be indistinguishable. In any event if no metastases are evident prompt surgery is indicated.

**Prognosis** This is decidedly poor. The operative mortality varies from 10 to 40 per cent. Hyman (1936) noted 90 per cent mortality in a follow up of 22 cases. Only 2 were living after five years and the majority died in the first year. Death is usually due to local recurrence metastasis cachexia or intercurrent infections. The sole ray of hope lies in earlier recognition with our improved diagnostic aids and more universal utilization of irradiation therapy.

**Treatment**—Surgical therapy depends upon the exclusion of metastatic foci and the functional capacity of the opposite kidney. If metastases are undemonstrable on physical examination and roentgen ray study, deep irradiation both pre and postoperatively is indicated. In general renal tumors are radio sensitive and a reduction in their size makes subsequent nephrectomy less difficult.



FIG. 937.—Wilms' tumor shows no barium pyelographic shadows and displacement of ureteral catheter. (Courtesy of Dr. S. R. Woodruff.)

Large tumors are best handled by the transperitoneal approach. This affords an opportunity to ligate the renal pedicle before manipulation of the kidney, thus avoiding possible dissemination of tumor cells into the blood stream. In addition, extension of the neoplasm into the renal vein and vena cava may be visible or palpable.

In cases of small tumors the lumbar route is applicable. Complete removal of the growth is essential for favorable prognosis. Because of the danger of hemorrhage, both real and potential transfusion should be considered as adjuvant therapy.

### PERINEPHRIC AND PARARENAL CONDITIONS

**Perinephric Infections**—Suppuration in the perinephric space in children is less common than in adults. The infective agent and route of infection is similar to that causing metastatic cortical

abscesses of the kidney (page 68a) Over 90 per cent of the cases are due to staphylococci with streptococci and colon bacilli accounting for the remainder

The hematogenous route directly or indirectly accounts for most infections Rupture of a cortical abscess through the renal capsule is quickly followed by infection of the perirenal fat Less common routes of extension are by way of the kidney liver pancreas duodenum appendix or pleura Trauma with urinary extravasation into the perinephric space is also a source of infection

**Symptomatology**—Fever chills anorexia gastro-intestinal upset acute loin pain or swelling or a protectively flexed lower extremity may lead one to suspect such an infection The urine may be clear In infants the diagnosis is especially difficult and is arrived at often by a process of exclusion Physical examination may reveal tenderness fulness or actual tumefaction in the renal area as demonstrated by bimanual palpation Point tenderness may be noted in the costovertebral angle together with a positive percussion (Murphy) sign

**Roentgenologic Findings** Roentgenographic studies may show a haziness of renal contour and obliteration of the normally sharp psoas muscle outline on the affected side In addition curvature of the vertebral column away from the site of the lesion is frequently noted It represents a protective mechanism just as the flexed thigh takes any stretch off the psoas muscle which underlies the suppurative area Aspiration with a long needle inserted in the costovertebral angle has been used as a diagnostic means The procedure is fraught with danger because the infection may be easily introduced into an unaffected kidney

**Treatment** This consists of lumbar incision as for any kidney exposure and drainage of the perirenal space If no evident pus pocket is found renal decapsulation should be performed in the search for cortical abscesses

The Adrenal Gland and its pathology is considered in conjunction with other endocrine glands (page 739)

**Pararenal Retroperitoneal Tumors**—While parietal retroperitoneal tumors do not properly belong in the urologic domain a diagnosis of their existence is frequently made during such investigation Clinically the presence of a large tumor in the loin or abdomen has led to a provisional diagnosis of kidney or adrenal tumor Radiographic studies may reveal normal configuration of the urinary pathways or secondary displacement or distortion of the organs by extrarenal masses Calcified areas or bone shadows may be discernible on the preliminary roentgen ray plate These tumors are usually of connective tissue or lymphatic origin The former include lipoma myoma combinations of such as chondromyxofibroma or sarcoma Lymphatic tumors are either chylous or

analogous to hygroma found in the cervical region. Unattached retroperitoneal tumors may be cystic, dermoid, teratomatous, sarcomatous or carcinomatous. Because the development and ultimate differentiation of male and female sex components take place retroperitoneally, gonadal or mesonephros maldevelopment with vestigial remains is a fertile source of such tumors. Campbell (1933) reported a pararenal teratoma in a child aged six months and noted 4 previous cases in the literature.

**Symptomatology** — This depends on the size of the tumor mass. Pressure may produce venous stasis (varicocele, leg or vulvar varicosities), backache, abdominal fullness, gastro-intestinal upsets, constipation or even renal colic due to ureteral displacement. Diagnosis is made by exclusion in the process of which an urologic investigation is essential and visualization of the intestinal tract with opaque roentgenographic media is optional.

**Treatment** — The surgical approach may be made posteriorly by loin or anteriorly by oblique inguinal incision. Radiation may prove of extreme value in the radio-sensitive lymphatic tumors with resultant remarkable reduction in size and simplification of the surgical problem.

### OPERATIVE TECHNIC

The usual approach for most renal surgery is by the lumbar route. By exposing the kidney through the loin practically all procedures on the organ, its pelvis and the upper ureter can be performed without entering the peritoneal cavity. Such an extra-peritoneal approach minimizes shock and the danger of peritonitis.

The commoner surgical measures comprise nephrectomy, resection or heminephrectomy, decapsulation, nephropexy, nephrotomy or nephrostomy, pyelotomy and pyeloplasty.

Certain necessary preliminaries must be considered prior to actual incision. For children the anesthesia of choice is ether preceded by atropine sulphate grain  $\frac{1}{60}$  hypodermically. In older subjects nitrous oxide or ethylene may be substituted. The preparation of the operative field is optional, some using 3% iodine, 5 per cent picric acid solution or 2 per cent mercurochrome in acetone.

**Kidney Posture** — Of utmost importance is the position of the patient. Since the kidney is to be exposed by incision in the ilio-costal space maximum advantage of the limited space available is essential. To this end the patient is placed in a lateral position and the gap between the lower costal margin and iliac crest exaggerated by elevation of a special kidney rest, breaking the table or by use of strategically placed sand bags. Failure to attend to this apparently insignificant detail represents the difference between ideal renal exposure and working conditions and constant operative difficulties.

**The Approach**—Lumbar incision is made to extend from the junction of the erector spinae muscle with the last rib downward and outward in hockey stick fashion to a point 1 inch above the anterior superior iliac spine. Some operators prefer a transverse incision starting at the costovertebral angle and extending anteriorly in the direction of the umbilicus. The direction and extent of the operative exposure will depend on the pathology at hand and its location.

Having made the skin incision superficial fat and fascia are incised and then in turn latissimus dorsi, external and internal oblique muscles and transversalis fascia. At this depth the ileohypogastric and ileoinguinal nerves traverse the parts and require retraction and preservation. The exposed pararenal fat is then cut and the perinephric space entered. The quadratus lumborum muscle may be partially cut or retracted. Sufficient working space is essential



tion of accessory vessels and respect for the vascular pedicle. Perirenal adhesions or a short pedicle may make the technical procedure somewhat more difficult.

**Resection or Heminephrectomy**—This may be indicated for localized suppuration, cystic disease, calculus or involvement of one segment of a double or horseshoe kidney. Mobilization having been effected, temporary vascular compression of the pedicle by the assistant's fingers or a rubber-covered clamp will minimize bleeding when the kidney substance is incised. Having resected, hemostasis is carried out by using mattress sutures buffered with fat pads obtained from the subcutaneous or perirenal areas. Muscle is an excellent hemostatic when tied in the incised area by sutures.

**Decapsulation**—The procedure is performed by making a small incision in the posterior capsular aspect of the convex border of the kidney. Passage of a grooved director under the capsule toward each pole and cutting the freed capsule over it minimizes trauma and bleeding. With thumb forceps or gauze the capsule can then be gently stripped as desired. The procedure is useful as a preliminary to nephropexy as well as to expose cortical suppurative lesions. At one time decapsulation was done frequently in cases of nephritis but has since been discarded as a therapeutic measure.

**Nephropexy**—This entails fixation of the kidney at a suitable level to correct undue mobility of the structure, kinking of the ureter and to promote drainage from the renal pelvis. Complete mobilization of the kidney and partial decapsulation to provide a raw area for adherence in a new bed constitute important steps. Fixation is secured by taking three mattress sutures through the upper, middle and lower portions of the renal parenchyma to anchor the organ within an area corresponding to the eleventh rib above and the costal margin below.

**Nephrotomy**—Incision through the renal substance permits of exploration of the parenchyma or pelvis and thus avoids pyelotomy. Calculi lodged in the kidney proper or in the pelvis may be removed in this way. Vascular pedicle compression during this procedure will save excessive blood loss and keep the operative field relatively dry. In cases where drainage is desired subsequent to the above steps or for relief of long standing obstruction in the kidney, a soft rubber catheter may be inserted into the pelvis through the nephrotomy wound and drawn out through the lumbar incision. Such a procedure has saved many kidneys which, with prolonged drainage, returned to a reasonable functional capacity.

**Pyelotomy**—Incision into the renal pelvis is usually made on the posterior aspect in the performance of pyelolithotomy. The ureter and pelvis lie posterior to the vascular pedicle and this anatomic fact is the basis for such an approach. The opening is made in the long axis of the tubular structure radiating from the hilum of the

kidney. Its size depends upon the proportions of the calculus. Such an opening may be used to insert a clamp through the renal substance and to draw a soft rubber catheter into the pelvis from without in order to accomplish nephrostomy drainage.

Two anchor sutures may be taken in the pelvis prior to incision or having incised the pelvis the edges may be grasped with Allis clamps. Curved forceps with suitable dentate jaws are used to pass through the pyelotomy wound to grasp the stones. Occasionally digital palpation may be necessary or advisable for localization of an elusive calculus or as a final check on its absence. Some urologists leave the pyelotomy wound open. We favor one or more sutures for loose apposition of the cut edges to promote healing by continuity and to avoid postoperative deformity. Suitable drainage is essential in closing the lumbar incision because of seepage of urine. One or two Penrose drains to the pyelotomy site suffice and should be left in until there is definite evidence of cessation of urinary

kidney. Since that time the anterior approach for such cases has been advocated and practised by many surgeons. Some urologists feel that ample exposure and minimal manipulation can be obtained by a transverse loin incision. Others avoid the procedure because of their unfamiliarity with and lack of temerity to do surgery in the upper abdomen.

A long upper rectus muscle incision is usually made and only in rare instances is it necessary to make an additional right angled extension to the wound. After the peritoneal cavity is entered the renal tumefaction is exposed by picking off the intestines. Some operators incise the posterior parietal peritoneum directly overlying the mass between small intestine (duodenum or jejunum) and colon (ascending or descending) and come directly upon the kidney and its pedicle. Others favor incision of the peritoneum in the lateral lumbar gutter with medial mobilization of the colon. The result affords excellent exposure of the vascular pedicle, which is ligated early, and the tumor mass is removed with as much perirenal fat fascia (Gerota's capsule) areolar tissue and ureter as may be deemed necessary. The renal bed may be extremely vascular and careful hemostasis is essential. The posterior peritoneal leaf is then sutured tightly and drainage effected by stab incision in the flank. Tumors of the adrenal may be exposed and removed in a similar manner the kidney being retracted downward during the operation.

## CHAPTER VIII

### THE URETER BLADDER AND URETHRA

### THE URETER

#### ANOMALIES OF THE URETER

MALFORMATIONS of the ureter while numerous assume clinical significance only when obstruction stasis infection or incontinence occurs.

**Duplication of the Ureter**—Abnormal splitting of the ureteral bud may give rise to incomplete or complete duplication and multiple ureters. As many as six ureters have been reported in one individual. Multiple ureteral orifices are frequent co-findings and may be located in or about the vesical trigone or ectopically in the bladder urethra genital or intestinal tract. Congenital alterations in the form of the ureter are productive of serious lesions in the child. Ureteral valves or valve-like spurs result in improper drainage. High insertion of the ureter into the renal pelvis occasionally creates a valve-like spur which by its interference with the normal emptying of the pelvis may necessitate resection with reimplantation of the ureter at the most dependent point of the pelvis. Uncorrected a progressive hydro- or pyonephrosis may eventuate. Presumptive diagnosis can be made by retrograde urography. Correction of the condition depends upon operative findings. Nephropexy may be indicated in association with an uretero-pelvic plastic procedure. Rarely is nephrectomy necessary or indicated unless more conservative means are inapplicable.

**Constriction of the Ureter**—Constriction may result from a variety of lesion. Spastic narrowings due to inflammation or mechanical irritation are often difficult to differentiate from an actual organic lesion. Serial roentgenography or repetition on different days may assist in accurate diagnosis.

Redundant mucosa of embryonic origin may by producing a valve effect cause obstructive hydronephrosis. Ectopia of the kidney in association with abnormally placed ureters may predispose to torsion or kinking of the ureter with subsequent obstruction. Similarly elongation and tonicity resulting from long standing obstruction or infection may produce numerous ureteral kinks.

**Partial Atresia**—Partial atresia of the ureter independent of infection represents a congenital anomaly comparable to such les-

ions in the biliary intestinal or lower urinary tracts. Sites of predilection are at the uretero vesical and uretero pelvic junctures and less commonly in the intervening ureteral segment.

Clinically such conditions may be characterized by dull or colicky pains, reflex gastro intestinal symptoms, palpable abdominal mass or persistent pyuria. Pathologically the process simulates that of an obstructive uropathy. (See page 646.) Hydroureter is soon augmented by hydronephrosis. The closer the obstruction to the kidney the earlier does hydronephrosis occur and the greater the parenchymal damage.

**Stricture of the Uretero pelvic Junction** may be due to intrinsic congenital narrowing or to extrinsic constriction from aberrant lower pole vessels. This topic has already been discussed in connection with renal anomalies. The roentgen ray is an invaluable aid in the localization and interpretation of the cause and effect of such lesions. Intravenous urography may reveal a large hydronephrotic pelvis representing apparently trapped contrast media. Retrograde pyelography will further confirm the degree of obstruction and vividly depict the actual constriction and filling defect in the shadow cast by the instilled radiopaque substance.

**Treatment**—Treatment depends upon the functional capacity of the involved kidney and also of its mate. Nephrostomy drainage may be instituted immediately and the obstructing bands or vessels cut with or without plastic repair of the uretero pelvic malformation. Strictures of the body of the ureter are uncommon and are most often associated with ureteritis or periureteritis and should be conservatively treated by repeated dilatation.

**Aberrant Pelvic Vessels** crossing that portion of the ureter lying between the brim of the bony pelvis and its termination in the bladder may produce marked constriction. (Fig 238.) Such extrinsic causes must be removed by resection and ligation of the bands.

**Megaloureter** independent of mechanical obstruction or neurogenic lesion is occasionally seen. (Figs 239 to 243.) The pathogenesis of the condition is not clear. Embryologic malformation, primary achalasia of the uretero vesical orifice with reflux and infection without visible obstruction have been held accountable for the condition by various observers.

Diagnosis is evident on cystoscopy and retrograde pyelography. A mechanical or neurogenic lesion should be excluded before considering the ureterectasis as a primary disease. Treatment may be unnecessary or may involve periodic lavage of the upper urinary tract. In extreme cases of infection uretero nephrectomy may be necessary.

**Lesions of the Ureteral Orifice** are few in children. Intramural stenosis occurs infrequently but its early recognition is essential to

spare the kidney from prolonged back pressure. We recently observed such an instance in a four months old child who unfortunately had a huge pyoureter and pyonephrosis above the point of constriction and an absence of the opposite kidney (figs. 242 and 243). Repeated and progressive dilatation of the stenosed portion of the ureter is indicated.

Ureterocele is often associated with stenosis of the ureteral orifice. The pathologic picture noted on cystoscopy is that of ballooning of the vesico-ureteral mucosa—a true herniation or cyst formation.

Treatment includes puncturing of the cyst, or dilatation of the ureteral orifice. Secondary changes in the upper urinary

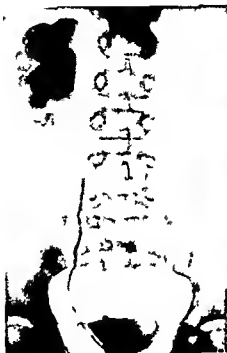




FIG 240—Cystogram of same child resulted in pyelography due to ureteral achalasia



FIG 241—Retrograde pyelogram case showing extent of obstruction due to intramural ureteral constriction to left of ureter represents barium in the colostomy segment



FIG 242—Roentgen ray taken ten minutes after previous pyelogram showing residuum of contrast medium and clear delineation of constricted ureter

tube in the female. No urogenital structure is exempt from anomalous implantation of the ureter and not infrequently the bladder, urachus, intestines or rectum mark the aberrant termination of the ureter.

From a clinical standpoint, the ureteral orifice may be so placed as to cause no symptoms and be noted only at autopsy, or infection or evident urinary obstruction may occur as a complicating factor. Thom in his series of 178 cases, reported 104 associated abnormali-

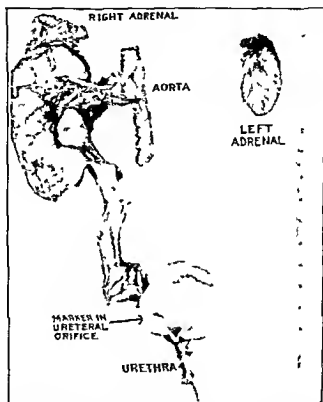


FIG. 243.—Postmortem specimen of same case showing solitary pyonephrotic kidney and pyoureter due to congenital stricture of the ureter. Despite absence of left kidney a normal adrenal was present.

ties of the upper urinary tract. The absence of symptoms accounts for the relatively low clinical incidence in the male. Ureteral ectopia in the female is frequently below the vesical sphincter in the urethra or vaginal vault, thus giving rise to persistent urinary incontinence.

In children incontinence associated with ectopic ureteral orifices has been occasionally mistaken for simple enuresis. Careful urologic study is necessary to prove the fallacy of such a diagnosis. A clinical diagnosis of persistent pyuria led Campbell (1937) to dis-



close ectopic ureteral orifices in 7 cases following complete urologic investigation

Diagnosis is made by intravenous injection of indigo-carmin and noting its appearance from a source other than in the voided or catheterized urine by excretory and retrograde urography and by cystoscopy. The nature of the anomaly and the extent of damage created by it is thus defined. Treatment depends on the nature of the lesion: diversion of the abnormal urinary stream and eradication of lesions secondary to the anomaly.

### INJURIES OF THE URETER

Injuries of the ureter seldom occur due to the protected position of the structure. In severe trauma to the kidney avulsion of the vascular pedicle may also include severance of the ureter at the uretero-pelvic juncture. Operative injuries are uncommon due to the infrequency of surgery on the ureter in children. Pelvic surgery in women contribute especially to such mishaps: ligation or severance occurring during the course of hysterectomy or adnexal operative interference. Perforation of the ureter by a ureteral catheter or bougie may occur following use of too rigid instruments or failure to respect inflammatory or congenital points of obstruction. The complication may pass unnoticed or manifest itself by periureteral extravasation of urine and suppuration. Suitable drainage should be instituted at once although mild cases may regress without active treatment.

*Obstructions* (page 646) *Infections* (page 682) and *Lithiasis* (page 666) of the ureter have been considered as urologic diseases and not dissociated as entities unrelated to similar lesions in the kidney above or the bladder and urethra below.

### TUMORS OF THE URETER

Tumors of the ureter and renal pelvis are of such rarity as to warrant only mention of the condition for the sake of completeness in considering their place in pediatric urology. Papilloma, myoma, fibroma and carcinoma have been reported in adults. Diagnosis at best is difficult and necessitates careful cystoscopy and retrograde pyelography. Surgery depends upon the nature and extent of the neoplasm and should always be completely radical.

### OPERATIONS ON THE URETER

*Operative Technique*—Surgical approach to the ureter varies with the location of the pathologic lesion. Operations upon the child's ureter are infrequent but may include ureterotomy, ureterectomy,

tures may be severed and the ureter freed of constrictions. Even lateral anastomosis of duplicated ureters, one of which is obstructed or has an ectopic orifice, has been reported.

**Transplantation**—Transplantation of the ureter to the bowel or skin (ureterostomy) is performed as a step in the surgical repair of extrophy of the bladder. The procedure is a formidable one and to Robert C. Coffey belongs the credit for its introduction and perfection. C. H. Mayo, Walters, Lunniss, Labey, Higgins and others have described modifications of the technic in recent years.

Exposure of the ureters and their implantation into the recto-sigmoid is effected transperitoneally. Labey (1935) described an extraperitoneal approach. After the ureter is isolated and mobilized irrespective of the method of exposure, a trough is made in the wall of the sigmoid by incision along one of the longitudinal bands. This forms a diagonal bed for the ureter and a puncture wound in the mucous membrane at the lower end of this incision permits introduction of the severed ureter into the intestinal lumen and creates a mechanical barrier to the reflux of liquid feces and gas into the ureter. The necrosing stitch operation of Higgins defers immediate severance of the ureter until a fistula has been produced between the ureter and intestinal lumen. Once this is established as noted by intravenous urography or the appearance of indigo carmine in the stool, the ureters may be severed and cystectomy performed.

## THE BLADDER

### ANOMALIES OF THE BLADDER

The bladder may be totally absent with the ureters opening externally or into the urethra. Extremely small (hypoplastic) or giant bladders (hyperplastic) represent rare occurrences. Double bladder is extremely rare although numerous instances of loculation, septum formation or diverticula have been misinterpreted as such. The condition is usually one of a series of associated defects and due to the absence of symptoms warrants no further attention.

### HERNIA OF THE BLADDER WALL

This may occur in association with inguinal, femoral or obturator herniae. The commonest type is that found with congenital inguinal hernia. At the operating table the hernia of the bladder may be the only organ involved or it may be attended by omental or intestinal protrusions. Of anatomic significance is the fact that the hernia of the bladder comes directly through the external ring and lies medial to the deep epigastric vessels and depending on the segment is or is not covered with peritoneum.

of the symphysis with distasis of the recti muscles and frequently inguinal hernie (Fig 244) Associated anomalies may include spina bifida hare-lip or club foot In the male epispadias and a rudimentary penis are the rule while in the female the clitoris is divided and the labia minora are separated anteriorly thus exposing the vaginal opening

**Prognosis** — This depends on the disposition of the case Untreated the patient pursues a truly tragic existence The exposed vesical mucosa becomes excoriated and bleeds readily Phosphatic incrustations may be deposited and several instances of malignancy



FIG 244 Intravenous urogram in an infant with exstrophy of the bladder Note appearance of dye in both renal pelvises and maldevelopment of pubic bones (Courtesy of Dr S R Woodruff)

in the chronically irritated mucosa have been reported Ascending infection represents the most serious hurdle that the victim has to surmount and about 50 per cent succumb before the age of ten

Walters (1932) summarizing the results of 76 cases at the Mayo Clinic in whom cystectomy was performed with transplantation of the ureters into the recto sigmoid reported a 3.9 per cent operative mortality Fifty nine of these cases were traced and known to be alive The fate of the remaining 17 is problematical Of the 59 living 27 have passed the fifth postoperative year and 13 have lived ten years Thirty or 50 per cent have no evidence of renal

infection. Such results which appear sporadically add hope for what was once a forelorn affliction.

**Treatment.** Treatment of exstrophy of the bladder is surgical. In the past numerous plastic operations were devised to re-form the bladder as a true viscus. Maydl tried transplantation of the trigone into the rectum but the results were poor. A more logical procedure was developed by Coffey to divert the urinary stream by transplantation of the ureters into the sigmoid with subsequent plastic surgery on the genitalia. This is followed by removal of the bladder and repair of the abdominal wall defect. The procedure will be described in the section dealing with Operative Technique.

Transplantation of the ureter into the bowel either after the method of Coffey or as modified by individual operators is now universally accepted and has proven efficacious. The principal danger of such a procedure lies in the development of peritonitis subsequent to the transplant. To obviate this Lahey (1935) has advocated an extraperitoneal implantation. Having passed the immediate postoperative stage an ever present hazard is the development of an ascending ureteral infection and ultimate renal insufficiency.

### THE URACHUS

This embryonal remnant of the allantois-vesical relation lip is occasionally the seat of anomaly or infection. It is considered at this point because of its intimate connection with the bladder. Normally the epithelial canal of the urachus is incompletely obliterated but the presence of the valve of Wutz prevents any regurgitation of urine. However if obstruction at the vesical neck or urethra creates sufficient intravesical hydrostatic pressure the normally obliterated canal may become patent and an umbilical urinary fistula result. In fact such an occurrence proved to be a life-saving measure in several cases with complete atresia of the urethra where no other path existed for the excretion of urine.

**Fistulae.** At times the urachus remains patent producing a true ordinary fistula. It may be patent only at its umbilical end with a constant or intermittent draining sinus frequently secondarily infected and occasionally leading to umbilical granuloma. Distinction should be made between it and a patent omphalomesenteric duct with fecal fistula. A patent urachus on the vesical side may simulate a diverticulum and is subject to the same complications—tumor, stone or infection. An unexplained pyuria may find its origin in the intermittent evacuation of such a sacculization.

**Cysts.** Urachal cysts result from the persistence of secreting epithelial cells. The result may be a small tumor mass situated just below the umbilicus. A cyst containing 20 liters was reported

by Almquist (1930) The contained fluid may be clear yellow as in a hydrocele, bloody, or frankly purulent

**Diagnosis** — The diagnosis of urachal lesions is made from physical examination, cystography and cystoscopy The finding of an umbilical or infra-umbilical tumor with or without umbilical drainage should make one suspicious Its superficial location and relation to the bladder, determined by cystoscopy and cystography, confirm the diagnosis

**Treatment** — This usually necessitates excision of the sinus or cyst Occasionally, use of a sclerotic agent may produce permanent obliteration In the presence of infection, incision and drainage should suffice and excision be deferred until a later date In effecting excision of the sinus or cyst, the injection of a dye such as methylene blue is a distinct aid in delineating the extent of the tract In the operative approach, a mid-line hypogastric incision can be used, encircling the umbilicus and when necessary, splitting the recti to expose the dome of the bladder The latter may be distended for easier identification Usually the umbilicus is excised and one should not hesitate to follow the tract down to the bladder, open the latter and excise the involved area of the wall The bladder wall can then be closed, leaving an indwelling urethral catheter in place, with suitable perivesical space drainage

## INJURIES OF THE BLADDER

In the child, the bladder lies high in the pelvis and when distended assumes almost the position of an intraperitoneal organ The tendency of children at play to disregard the call of Nature until absolutely necessary makes the viscus a vulnerable site for direct or indirect blows A fall, kick or crushing injury involving the hypogastric area may easily result in severe injury to the bladder The increase in vehicular traffic accidents in recent years makes such occurrences more common

**Pathology** — Trauma may result in contusion of the bladder wall, or rupture of the organ either intra- or extraperitoneally The rupture may be incomplete with slow seepage of urine followed by sudden onset of symptoms, or it may be marked with considerable perivesical extravasation

**Symptomatology** — One may find varying degrees of shock, local pain, inability to urinate, hematuria, lower abdominal rigidity or evidence of perivesical extravasation

**Diagnosis** — This may be made by careful physical examination, including rectal palpation, and the judicious use of the urethral catheter If no urine is obtained by catheterization, a few ounces of warm boric acid may be instilled Its failure to return confirms

ated by roentgen ray others are not radiopaque hence cystoscopic examination may be required to make the diagnosis

**Treatment**—This involves the removal of the foreign body and clearance of any infection that developed secondarily. The latter usually responds quickly to ordinary measures once the offending agent is removed. Many of the objects can be removed through the cystoscope using a flexible foreign body forceps or snare. The ingenuity of the urologist is tested in coping with such problems. Where the object is small enough to pass through the cystoscopic sheath the following procedure has served us on numerous occasions. With the instrument in the bladder the latter is well distended with irrigating fluid and the distal eye of the cystoscope is so shifted as to lie directly over and in line with the object. A sudden release of the vesical fluid by removal of the telescope from the sheath will often force the object forward through the instrument.

When the object is candle wax it may be disintegrated by using a solvent such as gasoline or carbon tetrachloride (with caution) for vesical instillation. Occasionally the size, nature or location of the foreign body may make an open operation the safest and most conservative therapeutic method. This is true particularly when severe cystitis, often phlegmonous or incrustation of the object with phosphatic salts has occurred. Suprapubic cystostomy is easily performed, the object is removed and temporary drainage afforded.

### OBSTRUCTIVE UROPATHY

Obstructive uropathy as it concerns the child's bladder has already been presented (page 640). Of special significance apart from organic mechanical lesions such as fibrosis and contracture of the vesical neck, hypertrophy of the trigone, hyperplastic mucosal folds or diverticulum is the problem of neurogenic vesical dysfunction (Fig. 245). It will be dealt with in a separate consideration of neurogenic lesions of the urinary tract (page 736).

Many of the vesical neck contractures may be successfully treated by repeated progressive urethral dilatation with bougies. Transurethral fulguration or resection of obstructing tissue has been effective in numerous instances while occasionally open operation with excision of a wedge of tissue from the sphincter floor may be necessary.

### CYSTITIS

Infections of the bladder are practically always secondary to upper or lower urinary tract suppuration. The bladder in its mid position is readily infected as a result. The subject has been presented in the general consideration of urinary tract infections (page 640).

## VESICAL LITHIASIS

Vesical lithiasis, a relatively common juvenile lesion, is one phase of urolithiasis and as such is included in a comprehensive approach to the disease (page 666)



FIG. 245 —Distended bladder due to hyperplastic vesical neck changes in a girl aged eight years

## TUMORS OF THE BLADDER

Their occurrence is very rare in childhood. Deming (1924) in a survey of primary vesical neoplasms in the first decade of life as seen by various American workers from 1912 to 1923 noted 3 infantile cases in over 2800 in all age groups. Beer (1930) found no children in a series of 500 personal cases of bladder tumor. Rabson (1935), in an exhaustive survey of the literature on bladder sarcoma found 42 cases in the first decade of life in a total of 202 collected cases. He observed a high incidence of teratomatous growths in children. Males were afflicted three times as often as females, 31 cases occurring in boys and 11 in girls.

**Pathology** —With the exception of the more common polypi or papillomata most of the growths are malignant and occur in the first five years of life. Sarcoma is more common than carcinoma while instances of myxoma, rhabdomyoma and fibroma have been reported. The site of the lesion is often trigonal with evidence of obstruction to the urinary flow. In females, the growth may

appear at the external urethral meatus. Infection is a frequent secondary complication.

**Symptomatology**—Pain, hematuria or urinary dysfunction is manifested by frequency, dysuria, urgency or tenesmus may be present. Obstruction to the outflow of urine is common with neoplasms in and about the sphincter and trigone. A tumor mass may be visible or palpable as an initial sign.

**Treatment**—This depends for its success on early diagnosis with coordinated use of roentgen ray or radium therapy, electrocautery and the surgeon's scalpel. The tumefaction may be first approached cystoscopically using high frequency current with deep roentgen ray or radium as adjuvant measures. Open operation and radical transvesical fulguration with or without resection of the bladder wall should not be delayed since herein lies the only salvation of the victim whose future at best is none too bright.

## OPERATIONS ON THE BLADDER

Because of the relatively high position of the bladder in children surgical approach to it is simple. When distended with fluid as a preliminary to its operative exposure the organ practically assumes an intra abdominal position. The various operations upon the bladder comprise suprapubic cystostomy, diverticulectomy, resection—subtotal or total plastic operations for extrophy and operative cystoscopy.

Inhalation anesthesia or for short procedures evipal is usually used. The patient is placed in the Trendelenburg position to facilitate an extraperitoneal approach by gravitation of the abdominal contents away from the operative field. A vertical hypogastric incision is made between the umbilicus and symphysis, its lowermost extent being at least one finger's breadth above the symphysis to preclude any secondary osteomyelitic infection of the pubic bone. The extent of the incision depends on the nature of the operation. If simple diversion of the urinary stream is desired little more than a stab wound is necessary, whereas for resection of the bladder wide mobilization of the structure must be effected.

The skin incision is deepened through the subcutaneous fat and fascia anterior rectus sheath, the rectus muscle retracted or bluntly split and the perivesical space exposed. Blunt dissection will reveal the bladder which has been previously distended with saline boric acid or metaphen 1 to 2000. The bladder wall is readily identified by its venous plexus. The peritoneal reflection is pushed back from the bladder wall but opening it during the process of mobilization is of no consequence if the injury is recognized and sutured at the time.

An especially simple cystostomy may be effected by using a small





FIG. 246 Postmortem urogram of a child with complete atresia of the bladder who lived only two hours. Note extreme degree of dilatation originating during intrauterine period. (Courtesy of D. S. R. Woodruff.)

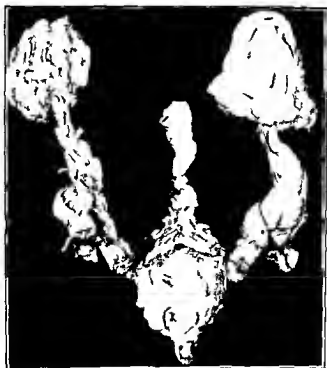


FIG. 247 —Autopsy specimen of same case. Note size of ureters and circumference of internal sphincter.

from the verumontanum to the external sphincter or posteriorly to the internal sphincter. The anomaly may assume the appearance of a finely perforated diaphragm of the iris type.

**Prognosis**—One should be guarded in determining the approximate life-expectancy of these patients. It is directly proportional to the extent of renal damage which in turn is dependent on the degree and duration of the obstructive uropathy. Usually the child presents himself for treatment when pathologic changes are so advanced as to reflect themselves constitutionally. Decompression of the over-



FIG. 248—Retrograde urogram showing marked obstructive phenomenon in a boy with posterior urethral valves. Note funnel shaped urethro-vesical shadow (Courtesy of Dr. S. R. Woodruff)

distended bladder and relief of obstruction produces transitory if not permanent improvement. The kidneys have displayed unusual recuperative powers in such cases.

**Treatment**—The child afflicted with obstructive urethral valves presents the same therapeutic problem as a victim of prostatism. Preliminary drainage either by an urethral indwelling catheter or suprapubic cystostomy is followed by cystoscopy at a later date after stabilization of the tract has occurred. It is dangerous to undertake immediate operative correction before establishing renal

balance measuring the degree of nitrogenous retention in the blood, and combating azotemia.

Transurethral instrumentation suffices in many cases. Fulguration or cutting may be done with the electrotome. Young has used his punch instrument on several cases. Occasionally the supra pubic or perineal approach may be undertaken.

**Hypertrophy of the Verumontanum**—This presents a similar picture to that of posterior urethral valves and must be differentiated from the latter condition (Fig 249). Engbee and Wollstein (1923) reported a series of 8 children with bilateral dilatation of the ureters, 7 of whom had hypertrophied verus without any demon-



Fig. 249—Urethro-cystographic delineation of hypertrophied verumontanum which caused partial urinary obstruction in a boy aged five years.

strable bands or valves. The age range was from three weeks to three and a half years with autopsy records in 7. Sections of the veru showed normal anatomic configuration with hypertrophy. An interesting observation was the dilatation of the urethra above the veru so that the latter almost takes an intravesical position, much as median prostatic hypertrophy produces. The symptoms and pathology are similar to the condition found in posterior valvular disease and the principles of treatment are likewise the same, with fulguration or resection of the obstructing mound of tissue.

**Congenital Diverticula, Cysts or Pouches**—These are rare and may arise from the urethral wall from anomalous ducts or from paraurethral glands. They assume clinical significance only when

they become infected, the site of calculus formation or create an obstruction. Kretschmer (1936) reported a case of urethral diverticulum and collected 20 instances from the literature. Diagnosis may be made on inspection when a soft cystic tumefaction lies on the ventral aspect of the urethral channel. Urethrography aids in delineating the exact site and extent of the pouch. Excision of the sac is often indicated and necessary.

Anomalous urethral channels vary from complete duplication to accessory channels. Our chief concern lies with hypospadias and epispadias.

**Hypospadias**—The condition represents an arrested stage of urethral development, with the urethra opening on the ventral aspect of the penis. This failure in the union of the genital folds in the male is an expression of hermaphroditic tendencies. In the female, a rare finding is that of a vaginal urethra, frequently termed hypospadias. The condition is often associated with other vaginal or vesical sphincter defects which require no attention unless sphincteric control is impaired, or childbirth results in complications at a later date.

In the male, the condition has been found approximately once in 350 cases on routine examination. The common types of the condition are balanic or glandular, penile and peno- or perineo-scrotal.

**Balanitic Type**—In this simplest variety the urethral opening lies in a line usually marked by the frenum. The site of the normal meatus is generally denoted by a dimpling of the glans, which is somewhat flattened in contour. The prepuce takes on the conformation of a hood above the glans. Treatment of these first-degree malformations can usually be omitted, since urinary function and procreation are unimpaired by the defect.

**The Penile Type**—This second-degree hypospadias is characterized by the appearance of the meatus anywhere along the ventral aspect of the shaft. There is usually a downward curvature of the penis and a folding of the lateral tissue so as to simulate two labia. In extreme cases, especially those bordering on the peno-scrotal type, one must distinguish between hypospadias and hermaphroditism. A groove usually extends forward from the meatus to mark the path normally traversed by the urethra.

**Peno-scrotal and Perineo-scrotal Types**—These are more exaggerated degrees of the anomaly. Undescended testes are occasional concomitant findings.

**Treatment**—The penile and peno-scrotal deformities require surgery. Apart from the mental aspect of the apparent deficiency is that of creating a normally functioning organ. Operative measures should not be undertaken until after the fourth year of life. Even then it is probably wiser to wait until some degree of coopera-

tion can be attained since at best the operative repair is delicate and much depends on keeping the field clean dry and untouched.

**Operative Technic** The principles include (1) The correction of the penile deformity and (2) reconstruction of the urethra. A transverse incision is first carried down to the corpora cavernosa in order to completely free the downward penile curvature by liberation of the shaft. Resuturing is done by making a vertical repair of the transverse wound or by filling the defect with skin graft.

The surgical attack on the urethra is best preceded by diversion of the urinary stream either by suprapubic cystostomy or external urethrotomy. Cecil feels that ligation of the urethra is the only proper means of obtaining a dry field for preservation of the reconstructed urethra until healing occurs. Many operators forego this preliminary diversion of the urinary stream but the risk of failure is far greater.

Operation for urethral reconstruction are numerous. For the balanitic type the urethra may be freely mobilized and implanted through a tunnel formed in the glans. Skin flaps with creation of an urethral floor have been variously applied by different surgeons. Duplex, Thiersch, Ombredanne, Beck, Mayo and Cecil are among those credited with specially devised techniques. Others have favored free transplantation of skin, mucous membrane, bladder appendix, ureter and heteroplastic urethral tissue.

Unless a surgeon has the proper outlook on this type of work which is most meticulous and trying, he should not undertake the care of the young patients who must be subjected to several stages of surgery. No rash promises can or should be made to parents as to the eventual outcome of one's surgical endeavor.

**Epispadias** The condition represents a congenital malformation of the urethra which opens on the upper aspect of the penis at a point behind its normal termination. The defect has been attributed to upward displacement of the cloacal membrane before the genital tubercle has formed.

The types of epispadias are similar to those of hypospadias, balanitic, penile or peno-pubic. The architectural defects are similar save that they involve the dorsal aspect of the urethra. Complete epispadias is usually associated with exstrophy of the bladder.

The condition is much less common than hypospadias but it is more frequently associated with urinary incontinence. In the female the defect may pass unnoticed or manifest itself by splitting or absence of the clitoris, involvement of the sphincter or any further defect of soft tissue or bone approaching the urethro-vesical defect of exstrophy of the bladder.

**Treatment**—The therapy of the obvious and impaired cases of epispadias embody the same principles and surgical approach as those presented for the correction of hypospadias. In the male

correction of the urinary and generative functions are indicated. In the female unless incontinence is present no operative measures are necessary. For incontinence plication of the urethra or transplantation of the pyramidalis or gracilis muscle fibers may be attempted. Failing in these ureteral transplantation may offer the only salvation for the child.

### INJURIES OF THE URETHRA

These are uncommon in early life. With severe trauma it is conceivable that the channel may be injured or severed and require immediate repair to prevent urinary extravasation. Local trauma may be self-inflicted by masturbation with harmful objects. However the child usually desists when local manipulation passes from the state of gratification to pain.

### FOREIGN BODIES IN THE URETHRA

These are occasionally found in the urethra in children who masturbate. The object used may get out of hand and slip into the deep urethra or bladder. Usually however no one is the wiser. The multiplicity of objects has been considered under Bladder Pathology (page 710). Recently one of us removed a cylindrical metal object trapped in the bulbous urethra of an adolescent boy. Attempts at transurethral manipulation failed because of inability of the operator to grasp the round object. External urethrotomy was done without sequela.

In the surgical treatment of stricture and following external urethrotomy it is wise to periodically check on the patency and adequacy of the urethral lumen. Failure to do so may result in secondary contracture often worse than the initial lesion.

### URETHRITIS

Infections of the urethra in the child are either (1) simple non-specific in nature due to local uncleanness or ascending involvement from balanoposthitis (2) secondary to upper urinary tract pathology or (3) specific due to the gonococcus.

Cleanliness and the correction of phimosis or paraphimosis by circumcision usually clears up the simple types of infection. Urethritis secondary to suppuration in the kidney or bladder demands eradication of the infection at its source.

Gonorrheal urethritis is a tragic occurrence in the infant. It is usually acquired by contact with an infected person or through the medium of articles of common usage—towels or bedclothes. In older boys direct contact may account for the infection.

Diagnosis, symptoms and treatment differ in no wise from the

infection in the adult. In the infant, however, local therapy is difficult if not impossible to carry out so that conservative palliative measures must be employed. In older boys, general measures as well as local irrigations and instillations with bactericidal agents are used routinely.

### URETHRAL CALCULI

These are very uncommon in children and occur only when some form of constriction or sacculation of the urethra is present. Occasionally a vesical calculus may become impacted in the posterior urethra with urinary obstruction. Instrumentation usually results in pushing the stone back into the bladder, where it can be handled as a vesical calculus (page 712).

### NEW GROWTHS OF THE URETHRA

Their occurrence is extremely rare. Epithelial hyperplasia with localized proliferation may produce polypoid or papillomatous growths which can be easily diathermized.

## CHAPTER XLIII

### THE MALE EXTERNAL GENITALIA

**Skin Affections** — While usually of no surgical significance, parasitic, pyogenic and non-specific lesions of the skin covering the external genitalia occur frequently either with or without significant pathology in the genito-urinary tract. Both sexes are equally susceptible to such afflictions.

Scabies is characterized by a papular, itchy eruption on the penis, scrotum or vulva in addition to its presence either in the axillary, gluteal folds or finger webs. A child sleeping with an infected adult falls an easy prey to the condition.

Pediculosis pubis is acquired similarly to scabies, although the absence of pubic hair until puberty makes the condition less annoying than in adults.

Fungus infection with the epidermophyton inguinale manifests itself by a genito-crural inflammatory lesion. Eczema and intertrigo are also common in infants.

Furunculosis, erysipelas and diphtheria may involve the external genitalia independently or in association with similar lesions elsewhere in the body.

Pruritus of the vulva and scrotum are usually associated with uncleanness or one of the above-mentioned skin lesions. Diabetes mellitus must, of course, be excluded.

Dermatitis venenata caused by poison oak or ivy may cause severe eruptions with systemic reaction, marked scrotal, penile or vulvar edema, and urinary difficulties. Cool compresses of subacetate of aluminum are usually effective.

Eczema, as part of the general disease, may be localized about the genitalia. Herpetic lesions may be due to uncleanness or occur as part of a *dermatitis medicamentosa* caused by iodides or bromides.

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## THE PENIS

### MALFORMATIONS OF THE PENIS

Malformations of the penis result from faulty development of the phallus, genital tubercle and urogenital sinus. They are frequently associated with urethral defects. Total absence of the



structure is extremely rare, Drury and Schwarzell (1935) finding 7 true cases in the literature since 1700 and adding 1 of their own. Associated congenital defects are common and ectopic erectile tissue has been noted frequently. The urethra either opens upon the perineum or into the rectum in such instances.

**Hypoplasia.**—Hypoplasia of varying degrees may occur and is usually associated with eunuchoidism, hypogenitalism or hermaphroditism. The penis may be concealed beneath pubic fat or a single covering of skin may enclose it and the scrotum.

**Hypertrophy.**—Hypertrophy of the penis is most often an expression of endocrine dysfunction.

**Adherent Penis.**—Adherent penis, resulting from the existence of a peno-scrotal web, is seen in hypospadias and the liberation of this downward curvature of the structure is the first step in its operative repair.

**Torsion.**—Torsion of the penis, or cleft formation may occur in hypospadias or epispadias as well as in cases of extrophy of the bladder.

### INJURIES OF THE PENIS.

Trauma to the penis is rare in the child unless due to accidental injury or inflicted with intent. Either through mere deviltry, for masturbation, or vexed by his nocturnal incontinence, the child may tie a string or wire about the shaft of the penis. Edema, gangrene and sloughing may eventuate if the constriction is tight enough and is not relieved early.

### INFECTIONS OF THE PENIS.

The penis is usually involved secondary to phimosis or paraphimosis. The bacterial organism is commonly the staphylococcus, streptococcus or colon bacillus, and responds to bathing and medicated wet dressings. Venereal lesions are rare.

**Tuberculosis.**—Tuberculosis as a complicating factor to ritual circumcision has already been referred to. Wolff (1921) collected 58 cases with a mortality of over 60 per cent in the first year.

**Diphtheria.**—Diphtheria of the penis has been reported following circumcision. In addition to this primary lesion, the infection may occur secondary to a nasopharyngeal focus. Treatment is the same as for diphtheria elsewhere in the body.

### TUMORS OF THE PENIS.

The only significant new growths in the child are congenital cysts. They are chiefly dermoid or mucoid in type and are limited to the median raphe. They arise from epithelial rests due to faulty closure of the raphe. Acquired epithelial cysts have

been reported following ritual circumcision and are attributed to epithelial inclusions as a result of inversion of the skin edges. These cysts are harmless and of no consequence unless infection, sinus formation or increased growth occurs. Surgical excision is then indicated.

## THE PREPUCE

Congenital adhesions of the prepuce to the glans or unusual narrowing of the preputial orifice represent the significant anomalies involving the prepuce. A short frenulum assumes importance later in life when it tears easily on erection and is a constant source of irritation.

Variations in the prepuce are often associated with penile malformations as in hypospadias and epispadias.

## CYSTS OF THE PREPUCE

Cysts of congenital origin, sebaceous or dermoid in character may occur on the prepuce or along the median raphe. Rarely do they require surgical excision.

## PHIMOSIS

The preputial orifice may be absent or almost pin point in size. While a certain degree is present in all new born males, the term true phimosis is applied when the orifice is so narrowed as to interfere with manual retraction of the prepuce behind the glans and with urination.

The prepuce may be adherent to the glans even though the orifice be adequate. Both conditions while benign may account for a multitude of symptoms some of which may assume serious proportions.

Obstructive phimosis with such narrowing of the orifice as to interfere with the normal egress of urine predisposes to preputial concretions of a soft or stony consistency and may also produce such urinary back pressure as to set up a picture simulating that of obstructive uropathy with bilateral hydronephrosis and renal insufficiency. In such cases urination is accompanied by balloon like distention of the prepuce and while the urine so trapped may dribble out the long standing obstruction and stasis has even resulted in death as reported by Urofino (1929) and others.

Inflammatory adhesions and smegma concretions under the prepuce may predispose to enuresis, irritable bladder, balanitis, balanoposthitis and masturbation.

**Treatment** — In certain sects, circumcision is a ritual and interestingly enough the study of cancer of the penis has yet to reveal a malignancy in one circumcised at birth. Some favor routine circumcision for all male children. If the nurse or mother pursues a regular routine of retracting the foreskin early in infancy, cleansing the glans and lubricating abraded surfaces, such an operation may be rendered unnecessary.

The physician may stretch the prepuce by opening the blades of a small forceps inserted through the orifice. Retraction may then be effected and continued by the mother. However such stretching may inflict as much trauma as to make circumcision a more conservative procedure. At best the division of the prepuce is extremely painful and should be preceded by a brief inhalation anesthesia preferably ethyl chloride.

Circumcision is indicated in the following conditions: (1) Congenitally narrowed preputial orifice, (2) adherent prepuce, (3) recurrent attacks of balanitis, (4) paraphimosis, (5) preputial calculi and (6) irritation of the glans giving rise to reflex stimuli (mictururbation).

Anesthesia in the new born may be attained by using a pacifier of sugar water, while general anesthesia is necessary in other children under twelve years of age. Local anesthesia comprising novocaine block at the base of the penis, is used in older cooperative boys.

**Circumcision** The technic while simple, may — and has been — terribly abused. The clamp method is best applied to the infant due to the smallness of the parts. The prepuce is first retracted behind the glans manually and all adhesions between skin and glans are severed. It may be necessary to stretch the orifice with a spread hemostat before complete retraction can be accomplished. A grooved director can then be used to separate the underlying adhesions. An essential to satisfactory circumcision is the complete retraction of the foreskin behind the glans so as to expose the entire circumference of the coronal sulcus. All inspissated detritus is then removed.

Having completely mobilized the prepuce a straight clamp is placed across the redundant portion, care being taken not to include the glans. A special slit plate may be substituted for the clamp. The excess skin is resected and the incised area is then inspected. It may be necessary to clamp and ligate an actively bleeding vessel either on the dorsal or frenular aspect. If the skin and mucous membrane are not well apposed two or three sutures of No. 00 plain catgut will facilitate healing and avoid deformity in the new born; however, suturing is usually unnecessary.

Special circumcision clamps are obtainable which may be efficiently employed. The principle is that of compression of the prepuce at the point of circumcision and subsequent guillotine

Compression usually obviates any necessity for ligation or apposition suturing. In the modern ritualistic circumcision the slit plate is used and suction is applied momentarily to the incision by means of an aspirating bulb. Pressure is applied for a few minutes and no further surgical procedures are essential.

The dressing in the infant is usually limited to liberal application of vaseline both as a covering protective against urine as well as a coagulant.

The *dorsal slit method* is applicable to older children and is carried out by applying two mosquito clamps on each side of the mid dorsal line and two at the frenum. Having freed the prepuce completely the two dorsal clamps are held up in one hand or by an assistant and a dorsal slit is made between them with straight scissors. The use of a grooved director as a protective measure is optional.

Having made the dorsal slit to within a few millimeters of the corona a guide suture is taken at the angle of the incision and the ends left long. Suture material is plain catgut the size varying from No. 000 to No. 0 depending upon the size of the patient. The redundant foreskin is then trimmed on either side starting from the previously applied frenular clamps. Active bleeding may occur from the frenular vessels or the dorsal veins. All such bleeding points should be clamped and ligated. The mucous membrane and skin are apposed with plain or mattress sutures at points on the circumference best localized as 12, 3, 6 and 9 o'clock. These sutures may be left long in older children and a strip of vaseline gauze may be tied within them so as to form a protective wreath about the incised area.

The *circular bloodless cuff method* is used by some but is less applicable to children. A circular incision is made in the skin and mucosa at the approximated points of amputation of the prepuce. The scalpel thus exposes the vessels and their prompt ligation before severance avoids unnecessary bleeding. The operation is more tedious than the clamp or dorsal slit technique. Irrespective of the technic one should always look for meatal constrictions and if present meatotomy should be performed at the same time.

**Postoperative Treatment** The dressings should be changed frequently to avoid unnecessary contamination. Any bleeding may be checked by pressure or by taking a mattress suture at the site of hemorrhage. Rarely does edema assume such proportion as to warrant cold compresses of magnesium sulphate or boric acid solution. Secondary contraction of the foreskin may follow a poorly done circumcision due to incomplete separation of adhesions or insufficient removal of mucous membrane. Such a secondary phimosis will necessitate further operative correction.

The error of removing too much skin may necessitate immobilization of the lax penile tissue by circular incisions. This corrects the extreme traction on the mucous membrane.

### PARAPHIMOSIS

Retraction of the prepuce behind the glans and inability to replace it forward over the glans is termed paraphimosis. The predisposing factor is a narrowed preputial orifice either congenital or inflammatory in origin. The condition is either created by a playful child or by the mother who fails to replace the retracted foreskin. A vicious circle is created by the narrowed retracted prepuce which impairs venous circulation to the glans. Penile edema results in further constriction behind the corona. If uncorrected, arterial embarrassment may occur resulting in subsequent gangrene.

**Treatment.** This may necessitate a dorsal slit with or without complete circumcision. If seen early, careful manipulation with preliminary lubrication of the glans may result in replacement of the prepuce to its forward position. In markedly inflamed paraphimosis a dorsal slit only should be done to relieve tension. Circumcision may be performed at a later date.

**Genital Edema** — Genital edema, often quite marked, occurs following breech presentation. Rarely does one see a marked edema of the genitals in male infants without apparent cause. The condition, which is to be differentiated from an inflammatory lesion, disappears rapidly and is thought to be lymphatic in origin.

### INJURIES OF THE SCROTUM

Injuries of the child's scrotum are rare due to its small size.

### TUMORS OF THE SCROTUM

Tumors, excluding intrascrotal ones, are rare in children. Cysts of sebaceous, dermoid or embryonic origin may occur. Cysts of the scrotal raphe represent persisting rests of the cloacal membrane. They seldom require surgical attention.

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## THE TESTIS

### MALFORMATIONS OF THE TESTIS

Malformations of the testis are referable either to its structure or abnormal location. Structurally, complete absence or agenesis of the glands is seen only in monstrosities. Absence of the one testicle, *monorchism*, has been reported, but this cannot be proven clinically since the organ may be hypoplastic or lie in an ectopic position. Supernumerary testes, *polyorchism*, are extremely uncommon and are usually noted with duplication of other genital structures. Cysts of the cord or *spermatocele* must not be confused with true duplication of the gland. Varying degrees of hypo- or hyperplasia of the testis may be noted especially accompanying endocrine dysfunctions. These anomalies of structure are merely of academic interest. More important is maldescent or malposition of the testicle.

**Cryptorchidism** — The condition embodies an arrest of the testicle in its descent into the scrotum. With the development and creation of the inguinal canal, combined intra-abdominal pressure, sphincteric action of the muscles forming the canal and guidance by the gubernaculum bring the gland through the external ring at approximately the seventh month of fetal life, and down into the scrotum at birth or shortly thereafter. Retention of the testis may occur (1) in the abdomen, (2) inguinal canal, or (3) pubo scrotal region. The most common type is the inguinal cryptorchid.

**Etiology** — Maldevelopment of the anatomic pathway traversed by the testis in its migration or hormonal deficiencies have been held accountable for the condition.

**Incidence**—Varying statistics have been presented showing an average occurrence of 1 case of cryptorchidism in every 40 boys under fourteen years of age while the condition is found approximately once in 250 men over twenty-one years of age.

**Intermittent retention of the testicle** occurs as a result of undue mobility of the gland and its gubernaculum in a spacious inguinal canal. The testis may assume an inguinal position for a time and then migrate to the scrotum. This can be promoted by taxis or by the application of warmth locally. Such a condition may lead to torsion of the cord and may have to be corrected surgically by anchoring the testis permanently in the scrotum.



FIG. 200—Boy aged ten years with Froehle's syndrome. Inguinal left testis. Note scrotal maldevelopment.



FIG. 201—Same patient after three months of anterior pituitary hormone therapy.

**Ectopic testes** have been reported in the perineal, crural, lower abdominal and penile areas. The perineal location is the most common, with the testis lying to one side of the median raphe above the anus.

**Diagnosis**—Diagnosis is usually evident on careful physical examination by palpation. The patient may be unaware of the defect or may seek medical attention due to some complication arising from the anomalous location of the organ.

**Complications**—Complications include congenital hernia, torsion of the spermatic cord, inflammation or trauma of the exposed gland or malignant degeneration. All of these have been shown to have a higher rate of incidence with respect to the undescended testicle than to one normally placed.

**Treatment** — Treatment of the undescended or ectopic testis is concerned with promotion of the migratory process and failing in this operative correction of the defect is indicated. The gonadotropic hormone from the anterior pituitary should be tried for a period of three to six months before undertaking surgery (Figs 200 and 201). One should not be too hasty in early surgical intervention although Bevan (1929) recommends orchidopexy between the ages of two and four years. He and others feel that adhesions are less marked and the elasticity of the tissues is greater in early childhood. Atrophy is said to parallel the length of time the testis lies in its aberrant position. For this reason if glandular therapy proves ineffectual after a fair trial and only a small proportion are responsive one should correct the condition surgically between the ages of eight and thirteen years. A factor which favors early repair is the frequency with which congenital inguinal hernia coexists. Some observers assert that it is always present. This fact and the possibility of complicating accidents lend weight to the argument of those favoring early intervention. Woodruff and Milbert (1936) reported a case of strangulation of an undescended testicle by a loop of omentum in a boy aged eight years. The occurrence is most unusual but attests to the advisability of surgery at the proper time.

**Orchidopexy** — Surgical fixation of the testicle in the scrotum may be accomplished by a variety of techniques. Brunzema (1929) compiled a list of some fifty different and often bizarre procedures listed for correction of the testicular maldescent or malposition. For simplicity we shall consider the two popular methods now in general use with some minor modifications only by individual operators.

The operation of Bevan comprises inguinal incision, complete liberation of the cord from its bed and placement of the testis in the scrotum. In this process the spermatic artery or vein may be sacrificed and the cord structures exposed retroperitoneally in order to obtain the necessary mobility to fix the gland in the scrotum without undue traction on the cord. A suture is taken through the gubernaculum testis and the base of the scrotum as well as a purse-string suture at the neck of the scrotum to counteract any retraction.

The Forek repair differs from the Bevan technic in its method of testicular fixation. After the testicle and cord have been completely liberated corresponding incisions are made in the scrotum and on the inner aspect of the thigh the testicle is drawn through the scrotal aperture and its gubernaculum is sutured to the fascia of the thigh. Thus scrotal-crural anastomosis assures against retraction and at the same time results in further development of the scrotum which is usually underdeveloped in cryptorchidism. A second-stage operation after an interval of two or three months



frees the attachment. While this operative technic is slightly more formidable than the simple orchidopexy, the excellent results merit the effort (Fig. 252).



FIG. 252. Torsion of bilateral undescended testes. Note size of left testis previously operated upon and scroto-crural anastomosis which will be separated in the final stage. (Case of Dr. S. R. Woodruff.)

**Torsion of the Testis.** The condition is actually a torsion of the cord and occurs uncommonly. However, failure to recognize its presence may result in needless sacrifice of the gland. A spacious inguinal canal coupled with a long gubernaculum or mesorchium usually accounts for mechanical interference with the circulation. The condition may recur and subside without sequelæ until unreheved torsion develops with subsequent testicular necrosis and atrophy.

**Symptomatology.**—The child may complain of severe local inguinal or scrotal pain and a swollen, tender mass be palpable. Hydrocele due to irritation of the tunica vaginalis is often present. Diagnosis must be made early since the testicle has been shown to be beyond repair in long-standing cases. Acute or subacute orchitis, strangulated hernia and mesenteric adenitis must be differentiated from the condition.

**Treatment.**—The treatment in early cases should be operative intervention with relief of the torsion and fixation of the testicle. In late cases, since the damage has been done, surgery may be deferred and the inflammatory reaction in the testis be allowed to subside with eventual atrophy of the gland.

**Torsion of the Hydatid of Morgagni** or appendix testis is similar to torsion of the cord both in principle and treatment. The condition is rare, is often confused with inflammatory orchitis or epididymitis and occurs before the age of puberty. Operation reveals the small tumefaction twisted on its pedicle and exhibiting hemorrhagic necrosis. Excision of the hydatid is necessary.

### INJURIES OF THE TESTICLE

Injuries to the testicle are uncommon. Dislocation or luxation, contusions or open wounds may result from external violence. Treatment depends upon the lesion and proper application of routine surgical measures. Tetanus antitoxin should be given in all suspected cases. Following trauma atrophy may occur as a result of local hemorrhage and pressure necrosis.

### INFECTIONS OF THE TESTIS

This seldom occurs in childhood and with the exception of frank tuberculous lesions surgery is not indicated. Acute orchitis has been observed in association with mumps, smallpox, osteomyelitis, scarlet fever and septicemia. The orchitis of mumps is interesting from the standpoint of future fertility. It is quite uncommon before the age of twelve years. Conservative measure with suitable cold or warm applications usually suffice. Rest in bed and strapping of the scrotum with adhesive plaster will promote resolution of the process.

**Tuberculosis**—Tuberculosis of the testis is less common than tuberculous epididymitis. Suppuration may occur early and necessitate incision and drainage or excision of the diseased area. The condition is usually chronic typified by cold abscess in contradistinction to the hot painful pyogenic infections.

**Luetic Orchitis**—Luetic orchitis may occur as a part of systemic congenital syphilis. It is characterized by the presence, often bilateral, of a hard painless swelling with or without epididymal involvement. Adequate antiluetic therapy usually produces an appreciable resolution of the process.

### TUMORS OF THE TESTICLE

Testicular tumors are extremely rare. The few cases cited in the literature include mixed tumors with inclusion rests of extragenital tissue (bone, cartilage and mucous membrane), teratomata and sarcomata. The latter are highly malignant tumors, ulcerate and metastasize rapidly and result in early death. They are usually of the spindle- or round cell type. Deep roentgen ray therapy and radical surgery offer the only proper attack on a practically hopeless disease.

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FIG. 252.—Török repair of bilateral undescended testes. Note size of left testis, previously operated upon, and scroto-crural anastomosis which will be separated in the final stage. (Case of Dr S R Woodruff)

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**The Prostate — Hypoplasia** — Hypoplasia of the gland may occur as part of general endocrine dysfunction. Congenital cysts may assume such size as to interfere with urinary flow. They either rupture spontaneously or must be incised or bluntly punctured. Injuries and infections are rare.

**Sarcoma of the prostate**, though uncommon, occurs most often in children under ten years of age. Young (1918) cited 35 cases collected from the literature, including 1 of his own. Fifteen patients were under ten years of age. Similarly, Sysak (1924) reported on 22 cases of sarcoma of the prostate in children between six months and nine years of age. Pathologically, one notes a variety of cell types, chiefly spindle cell or lymphoid in character.

The diagnosis is usually evident since the hidden nature of the lesion and the slow growth and extension of the tumor mass makes for delay in seeking medical care. Treatment, though usually unavailing, should include intensive roentgen-ray and radium therapy. Surgery alone offers little promise of success.

**The Epididymis, Vas Deferens and Spermatic Cord** — These structures are relatively free of disease in childhood. Pyogenic or tuberculous infections may involve the epididymis as a secondary manifestation. Palliative measures or, rarely, incision and drainage will suffice for pyogenic infections, while epididymectomy is indicated for localized tuberculosis in the structure. Spermatocele has not been reported before the age of puberty.

**Varicocele**, or dilatation of the veins constituting the plexus, is of two types:

1 Symptomatic—secondary to interference with spermatic vein drainage due to tumefactions of the kidney or any other structure overlying the vessels.

2 Idiopathic—without any definite etiology.

Burney (1910) reviewed 403 cases and found 8 per cent had varicocele since birth and with definite familial tendencies. The characteristic find is a coiled aggregate of scrotal veins with disappearance of the tumefaction when the child assumes a recumbent position.

Treatment of idiopathic varicocele in the child is non-surgical if no symptoms are present. The symptomatic type merits determined effort to elicit the cause of the condition.

**Hydrocele and cysts of the cord** are to be differentiated from true hydrocele, although their treatment is the same.

Diseases of the seminal vesicles are of no clinical significance in children.

The verumontanum has already been cited (page 718) as a source of urinary obstruction through congenital benign hypertrophy.

## HYDROCELE OF THE TUNICA VAGINALIS.

This may occur as a symptomatic or idiopathic manifestation. The former may follow trauma, as in birth or due to a fall or kick, or after an acute inflammatory process in the testicle or epididymis. The idiopathic type needs suitable explanation. The congenital form, often seen in infancy, usually disappears spontaneously.

Accumulation of fluid in any portion of the processus vaginalis may result in a variety of clinical and pathologic types:

1 Vaginal type, with fluid accumulation in the tunica vaginalis. This represents the commonest form.

2 Congenital or intermittent type, with communication with the peritoneal cavity, resulting in variations in size of the tumefaction and its disappearance on lying down.

3 Infantile type, in which there is no communication with the abdominal cavity.

4 Encysted hydrocele of the cord, with fluid accumulation in one unobliterated portion of the tunica. A round swelling may be noted independent of the testicle or epididymis.

**Diagnosis.**—This is usually easy. The hydrocele mass is cystic and transmits light in contradistinction to hematocele or spermatocele in which the medium is too dense to permit transillumination.

**Treatment.**—Treatment depends upon the age of the child, the size of the mass and the absence of any primary condition to which the fluid effusion is but an irritative response. In infants, the swelling frequently disappears spontaneously. The tumefaction may be aspirated with or without injection of a sclerotic agent, or it may be removed by open operation. The "bottle" operation consists of incision of the hydrocele and eversion of the sac. The von Bergman procedure involves excision of the sac down to its testicular reflection. In children the former is usually preferable since the latter is productive of more local reaction. Any accompanying hernia may be repaired at the same time and the processus vaginalis should be freed and resected to the internal inguinal ring.

## HEMATOCELE.

Hematocele represents an effusion of blood into the tunica vaginalis. *It resembles simple hydrocele and its treatment, if it fails to resolve spontaneously, is the same.* Trauma most often accounts for the condition.

## DISEASES OF THE ACCESSORY SEX GLANDS AND SEMINAL TRACT.

Male children are little affected by the numerous lesions of the genital tract which befall their elders. Injuries occur very infrequently and then only when severe trauma has been incurred by the surrounding soft tissues or osseous structures. Infections are seldom



primary and when lodged in the epididymis or prostate represent hematogenous coccus or tuberculous foci or ascending infection by the gonococcus

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## CHAPTER XLIV.

### NEUROGENIC DISEASES

INVOLVEMENT of the brain, spinal cord or peripheral nerves may produce significant changes in the urinary tract. The subject is still in the stage of development and much of the fact and fancy concerning ureteral, vesical and sphincter innervation through peripheral pathways requires crystallization.

Without considering the complexities of the neurologic aspect, the effect on the urinary tract is noteworthy. Adynamic obstruction with stasis and infection usually occur. Clinically, acute or chronic urinary retention is noted, or incontinence of the paradoxical overflow type is present. It is important, of course, before consigning a case to the neurogenic category, to rule out any demonstrable organic pathology by complete urologic investigation.

Manifestations of the central nervous system and spinal cord resulting in paralytic or atonic bladders in children include: (1) Fractures and dislocations, with attendant hemorrhage or nerve tissue destruction (birth injuries are included in this group); (2) benign or malignant tumors of brain and cord; (3) meningocoele; (4) acute infections (cerebrospinal meningitis); (5) syringomyelia; (6) transverse myelitis, and (7) polio-myelitis.

*Peripheral nerve involvement* and secondary bladder changes may occur with injury to the sacral nerves, either by direct trauma or through association with spina bifida or other lumbosacral anomalies.

*Infectious ferens*, including diphtheria, measles, scarlet, and pneumonia have been held accountable for urinary dysfunction. A larger group without evident etiology or neurogenic pathology has been the subject of considerable speculation. Whether spina bifida or similar lumbosacral defects produces peripheral nerve involvement is a debatable subject. This is especially true if no other evidences of peripheral sensory or motor changes can be found. Neurosurgery has been performed in such cases with conflicting reports both as to pathology and results. In other cases prosternal denervation has been done.

Beer (1930) feels that many of these cases have as their basis contraction of the vesical neck due to disharmony between sphincter muscle and detrusor. Because of the uncertainty existing as to the true causative nature, it would seem that a conservative surgical

policy in treatment should be pursued. The urologist usually sees the child late in the disease process and his problem generally entails combating infection or establishing suitable drainage. In extreme cases cystostomy, nephrostomy or uretero-nephrectomy may be necessary. A word of caution is pertinent in this connection. Both upper urinary tracts should be carefully studied before undertaking surgery, since bilateral involvement is not uncommon. A poorly functioning organ in such instances is better than none at all. Cystoscopy, urography and cystometry may prove valuable aids in proper evaluation of the individual case. The prognosis is routinely poor.

**Enuresis**—Closely linked to neurogenic dysfunction of the urinary tract is the symptom of enuresis. From the surgical viewpoint the condition assumes significance only as a possible expression of underlying pathology somewhere in the urinary tract.

**Etiology** The etiology of enuresis *per se* is debatable. Psychic and neuropathic factors, habit and faulty training, depth of sleep and the influence of masturbation have all been expounded. The multiplicity of lines of therapy attests to the far from settled etiology.

From an urologic standpoint, enuresis which persists after the age of four years despite medical therapy, and cases which develop in later childhood after a period of urinary continence merit complete urologic study. While over 90 per cent of the cases may show no pathology, the occasional case of pyonephrosis, tuberculosis or anomaly with or without obstruction makes the expense and effort worth while.

The study should include determination of bladder capacity and residual urine, bacteriologic investigation of the urine, cystoscopy and urography including cystography. Examination of the external genitalia may reveal stenosis of the external meatus of the urethra or phimosis with considerable irritating detritus underneath the prepuce. Correction of such simple lesions which produce reflex irritative phenomena may result in cessation of the enuresis. Instrumentation by its dilating effect may similarly result in cure. We have found that mothers are only too willing to submit children for complete study in an effort to correct any annoying conditions.

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## THE ENDOCRINE GLANDS IN UROLOGY

The endocrines play a prominent role in the development of primary and secondary sex characteristics in the child. While the subject is far from clarified, we can set down certain clinical syndromes which have been rather clearly delineated.

## THE GONADS

Prepubertal deficiency manifests itself in eunuchoidism. Certain cases of cryptorchidism exhibit this trait while a more classical example is the Skoepe. On the other hand prepubertal hyperactivity expresses itself in sexual precocity. In addition to the influence of the testis and its proper secretions disturbances of the adrenal, pineal and thyroid glands are responsible for a series of anomalous sexual developments.

## THE PITUITARY GLAND

Dysfunction is manifested by hyper- or hyposecretion of its anterior lobe. Acromegaly or gigantism results from hyperpituitarism but since these occur after puberty we shall confine the discussion to infantilism produced by hypopituitarism. Development both general and sexual is arrested. Froelich (1901) first described the type—male obesity, most pronounced on the trunk, breasts, pubis and hips; pubic and axillary hair absent, and the external genitalia infantile. Frequently the testes are undescended and the scrotum much reduced in size. Such a clinical condition is referred to as Froelich's syndrome or dystrophia adiposogenitalis. Infantilism may also be associated with hypofunction of the thyroid gland as in *cachexia strumipriva* or cretinism.

**Hypogonadism.**—Hypogonadism and cryptorchidism are closely bound to the pituitary. The gonadotrophic hormone present in maternal and fetal blood and in the early postnatal period stimulates testicular development and migration. Ingle (1932) made a real contribution to the subject by his work on animals. Gordon (1936) in a study of 519 boys with hypogonadism or cryptorchidism found associated endocrine maladjustment twice as frequently as in children without thyroid or pituitary deficiencies.

Study of subjects with hypogonadism or cryptorchidism of the endocrine type has shown definite clinical evidence of thyroid-pituitary deficiency. According to Gordon this is manifested in a low basal metabolic rate, low specific dynamic action of proteins and high blood values of cholesterol, chlorides and uric acid.

**Treatment.** Treatment of hypogonadism is based on supplementary glandular therapy of thyroid and anterior pituitary extracts orally or hypodermatically. Cryptorchidism which does not respond after a reasonable period (six months) of endocrine therapy is undoubtedly due to mechanical interference with testicular descent. This subject is considered under *Surgery of the Testicle* (page 731).

### THE PINEAL GLAND

Secretion of the pineal gland which plays an important part in the early years of the child's existence acts antagonistically to the pituitary gland. It is believed that the pineal body represses premature development of the sexual organs. Tumors of the gland apart from cerebral manifestations often cause an abnormal body development between the ages of four and eight years. There is an early growth of hair on the pubis, obesity, marked enlargement of the penis and less often of the testes and breasts. The voice may also change. This syndrome is called *macrogenitosomia præcox*. Pineal extract is of questionable value. Deep roentgen ray therapy and surgery offer the most effective means at our disposal.

### THE ADRENALS

Although the anatomic and histologic structure of the adrenal has been well defined, one cannot make a similar statement with respect to the physiology of the gland. Numerous cases of precocity, pseudohermaphroditism and hirsutism have been attributed to hyperactivity of the cortical cells, while their hypofunction has been held accountable for certain types of obesity, amenorrhea, infantilism or senility in early life. Sudden rises in temperature may be caused by hyperactivity of the medullary cells or in turn hypofunction of the cortical cells.

While tumors of the adrenal gland represent our chief concern in dealing with surgical aspects of the gland, several clinical entities will be listed briefly. Addison's disease or more truly adrenal insufficiency is rare in children. However the condition may occur as a result of intoxications, burns or adrenal hemorrhage of traumatic or thrombotic origin. Clinically collapse and hyperpyrexia occur but the diagnosis is most often made postmortem. Rabinowitz (1923) reported 2 cases of hemorrhage into the adrenal in babies of seventeen and eighteen months respectively, while Hamill (1901) collected 90 cases occurring in new borns.

**Tumors of the Adrenal**—Tumors of the adrenal are relatively rare and unless they manifest themselves by their size or by secondary sex disturbances frequently pass unrecognized until death. Our chief concern lies with the highly malignant medullary tumors found in children. Connective tissue tumors are extremely rare, are usually found on postmortem examination and have little clinical significance. They include fibroma, lipoma, myoma, angioma and sarcoma.

Cortical tumors, either adenoma, carcinoma or melanoma, are often characterized by genito-adrenal syndromes. They occur in adults and less often in children. The genito-adrenal syndrome

## PART IX

# NEUROLOGIC SURGERY

By JOHN E. SCARFF B.S. M.D.

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### CHAPTER XLV

### HYDROCEPHALUS

HYDROCEPHALUS is not a discrete pathologic clinical entity but rather a physical state which follows interference with the normal circulation of the cerebrospinal fluid.

Obstructive and non-obstructive types occur. These may be produced by lesions as diverse in nature as (1) congenital anomalies (2) inflammatory processes with their resultant cicatrices and (3) expanding lesions such as tumors, cysts and abscesses.

The condition creates two distinct syndromes. (1) The familiar frank hydrocephalus of infancy in which the whole head enlarges disproportionately and (2) the less well known but equally important occult hydrocephalus of older children in which the head does not become unduly large and the symptoms of increased intracranial pressure are the dominant clinical features.

One basic pathologic factor is common to all cases of hydrocephalus namely interference with the circulation and absorption of the cerebrospinal fluid. Knowledge of the normal circulation is accordingly of prime importance.

**The Spinal Fluid Circulation**—The cerebrospinal fluid is formed by the choroid plexuses which lie within the ventricles of the brain principally the two lateral cerebral ventricles. From the latter the fluid escapes by way of the two foramina of Monroe into the narrow slit like third ventricle situated in the mid line between the optic thalami (Fig. 253). It then passes through the aqueduct of Sylvius into the fourth ventricle which lies in the posterior cranial fossa between the cerebellum above and the medulla oblongata below. Thus far the fluid has been on the inside of the brain in spaces lined by ependyma. From here however the fluid passes through the foramina of Magendie and Luschka into the cisterna magna which lies outside of the brain. This cisterna is in reality a dilated part of the pia arachnoid or subarachnoid space which entirely surrounds the brain and spinal cord.

From the cisterna magna the cerebrospinal fluid is distributed caudad within the spinal subarachnoid as far as the lumbar sac and cephalad by channels encircling the brain stem and pons to other large subarachnoid spaces lying ventrally along the base of the brain—the cisterna interpeduncularis and the cisterna intercrurata. From these cisternae the fluid moves upward over the surface of the cerebral hemispheres following in a general way the

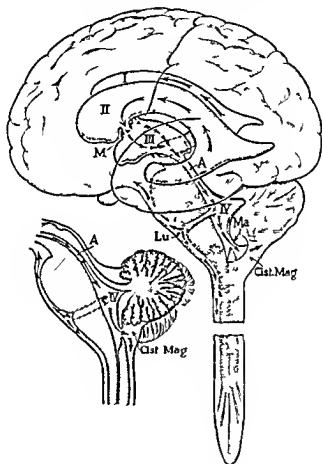


FIG. 13. Schematic drawing showing circulation of the spinal fluid.

course of the arterial tree. The fluid spaces are macroscopic within the limits of the sulci but over the top of gyri are only capillary in thickness.

Absorption of the cerebrospinal fluid eventually takes place from the small capillary spaces overlying the cortex by a mechanism not completely agreed upon. Certain investigators contend that the fluid passes from the subarachnoid system into the superior longi-

tudinal sinus through the active agency of the Pacchionian granulations, while others believe the fluid reenters the blood stream directly from the depths of the cerebral sulci.

Interference with the normal circulation and absorption of the cerebrospinal fluid produces an increased intraventricular fluid pressure. This distends the ventricles and compresses their walls which in reality are composed of the cerebral cortex. The cortical walls thereupon undergo "pressure atrophy" and become progressively thinner, while at their expense the ventricular cavities become progressively larger. Hydrocephalus is thus a physical consequence of some other primary pathologic process, and is analogous in many respects to hydronephrosis. The process may continue until the cerebral cortex in places is only a few millimeters in thickness and almost the entire intracranial space is filled with fluid. Hence the term "hydrocephalus" which means "water on the head."

### OBSTRUCTIVE HYDROCEPHALUS

Obstructive hydrocephalus, as the name implies, is caused by mechanical interference with the free circulation of the cerebrospinal fluid within the brain, at some point between its origin within the lateral cerebral ventricles and its escape into the subarachnoid system by way of the foramina of Luschka and Magendie. Obstruction is most likely to occur at those points where the cerebrospinal fluid channel is narrowest, *i. e.*, at the foramina of Monroe, in the third ventricle, at the aqueduct of Sylvius, in the fourth ventricle, or at the foramina of Luschka and Magendie. Dilatation occurs only in those parts of the ventricular system proximal to the obstruction.

**Etiology**—Various pathologic processes may cause obstructive hydrocephalus such as congenital atresia, septic and non-septic inflammatory processes and their resultant adhesions, embryonal cysts, abscesses, and tumors. (Figs 254 to 257.) The so-called "congenital idiopathic" cases of obstructive hydrocephalus result from congenital atresia (or insufficiency) of the aqueduct of Sylvius, but in the writer's experience this is a comparatively infrequent cause of the obstructive hydrocephalus even in very young infants.

*Tumors, cysts and abscesses of the cerebellum are a common cause of obstructive hydrocephalus in children.* Congenital cysts of the pituitary anlage are also frequent during this period and often produce obstruction through compression and closure of the foramina of Monroe. Neoplasms within the third ventricle and those arising from the pineal body may also occasionally produce obstructive hydrocephalus. Finally, there is a form which follows inflammatory reactions, either septic or non-septic, of the pia arachnoid spaces or membranes, which results in cicatricial closure of the foramina of Luschka and Magendie. In such cases the cerebrospinal



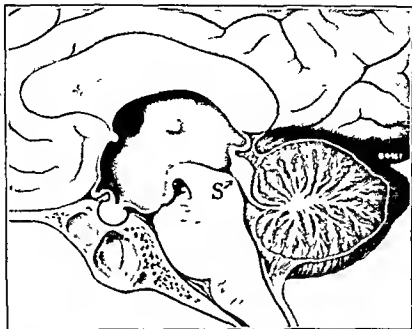


FIG. 254 —Obstructive type of hydrocephalus. Congenital atresia or insufficiency of the aqueduct of Sylvius.

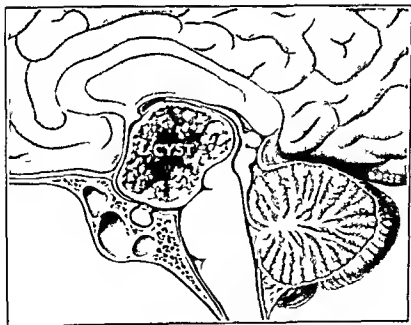


FIG. 255 —Congenital cyst (Rathke's pouch) of the pituitary anlage.

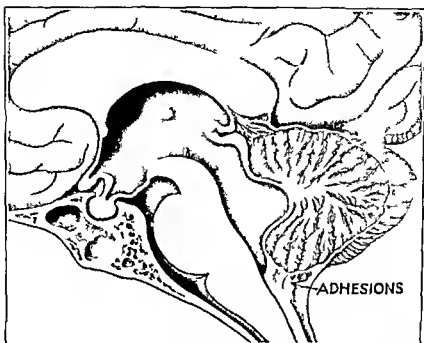


FIG 266 — Adhesions of the pia arachnoid along the foramina of Magendie and Luschka

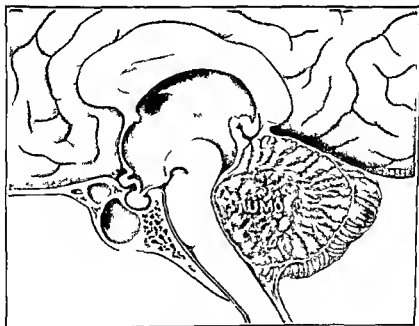


FIG 267 — Tumor of cerebellum

fluid cannot escape from the fourth ventricle into the cisterna magna and pia arachnoid spaces, and obstructive hydrocephalus follows (Fig 258)

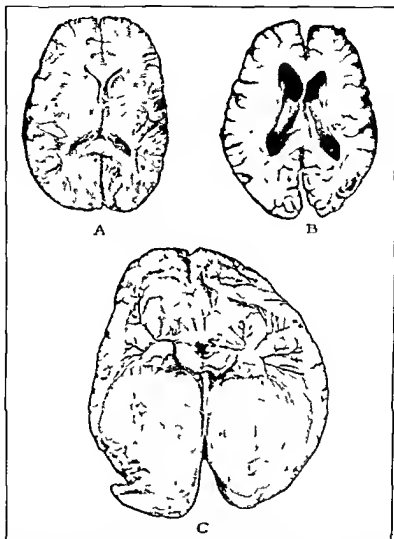


FIG 258 —The Cerebral Ventricles A in a normal brain B in the brain of a child developing hydrocephalus after ossification of the cranial sutures (occult hydrocephalus) C, in a hydrocephalic infant a few months old

### NON-OBSTRUCTIVE OR COMMUNICATING HYDROCEPHALUS

The non-obstructive or communicating type of hydrocephalus, in contrast to the obstructive type, is generally idiopathic in origin. In such cases there is no apparent mechanical interference with the

free circulation of the cerebrospinal fluid and no anatomic explanation for the increased pressure. Apparently the cerebrospinal fluid cannot be absorbed from the *pia arachnoid* (subarachnoid) spaces overlying the cerebral cortex as rapidly as it is formed within the ventricles by the various choroid plexuses. Whether this is due to an increased production of fluid or a decreased absorption is seldom determinable. In rare cases where non-obstructive hydrocephalus develops after a child has recovered from meningitis it seems probable that the postinflammatory cicatrices obliterate parts of the subarachnoid spaces overlying the cortex and in this way reduce the normal rate of absorption.

**Clinical Types**—The clinical picture presented by hydrocephalus in the very young infant is a familiar one. Since the bones and sutures of the skull have not completely ossified, the entire head yields to the increased intracranial pressure, and enlarges. This enlargement follows a certain pattern—it is limited almost entirely to the vault and the face remains essentially normal in size. The fontanelles remain open and bulge tensely, the sutures are separated, the frontal bosses are unnaturally pronounced, the eyes appear to be pushed downward, and the palpebral fissures become narrowed and almond shaped. The head may reach an enormous size (Fig 259). But in spite of this, and possibly because of it the signs of acute intracranial pressure, such as headache, nausea, vomiting, blindness and coma, are ordinarily lacking. The optic discs usually undergo primary atrophy, but only rarely show papilloedema. Pressure decubiti of the scalp become increasingly difficult to prevent. Death, in untreated cases, eventually comes as a result of marasmus and intercurrent infection.

In older children, whose cranial sutures have already ossified, the symptoms and signs of hydrocephalus are quite different from those in infancy. The ventricles of the brain become progressively distended by the increased pressure of the fluid within them and may attain great size at the expense of the cortical substance which they replace, but the skull itself does not enlarge. This may be designated as "occult" hydrocephalus. If the process is not arrested, there eventually develop the classical signs of acute intracranial pressure—headache, nausea, vomiting, edema of the optic nerve with progressive blindness, stupor, and finally respiratory failure (Fig 260).

**Diagnosis of Hydrocephalus**—This should comprise not only the recognition of the disorder but also identification of the type, *i. e.*, obstructive or non-obstructive, and a determination of the etiology.

Hydrocephalus which occurs before ossification of the skull has been completed is readily revealed by the characteristic enlargement of the head. The "occult" type, however, which develops after the bones and sutures of the skull have completely ossified, is at times more difficult to recognize. It should always be suspected

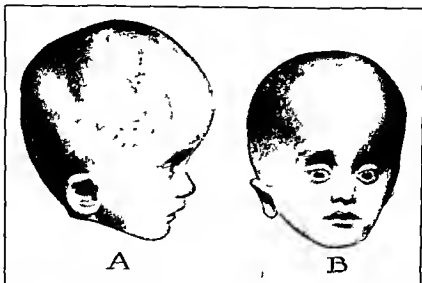


FIG. 259—Characteristic head changes in Infantile Hydrocephalus. A Disproportionate enlargement of the cranial vault prominent and overhanging frontal bossæ and pushed down appearance of the eyes. B fontanelles widely open bulging and tense.

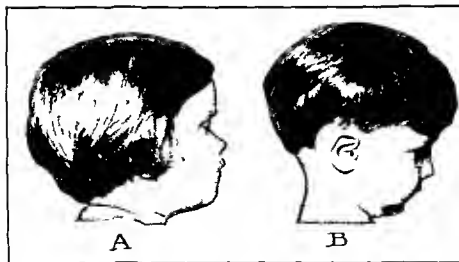


FIG. 260—Both these children have hydrocephalus with increased cerebral fluid pressure dilated ventricles and cortical atrophy. Their heads are not enlarged however because the sutures had ossified before the disease started. Hydrocephalus in A was caused by a benign congenital cyst of the cerebellum and in B through closure of the foramina of Magendie and Luschka by scar tissue following non-suppurative adhesive arachnoiditis. Both are symptom free three years following operation.

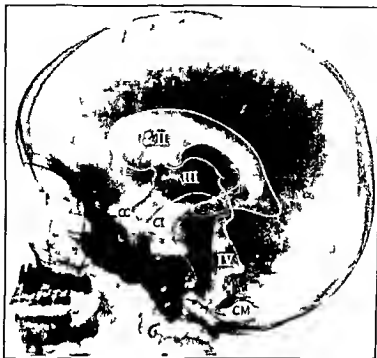


FIG. 961 — Lateral and A P Encephalograms of a normal brain. I and II indicate the lateral cerebral ventricles III and IV the third and fourth ventricles CC optic chiasm CI optic chiasm CM optic chiasm

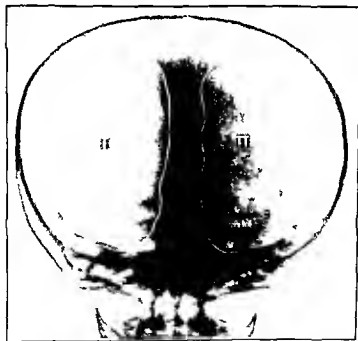
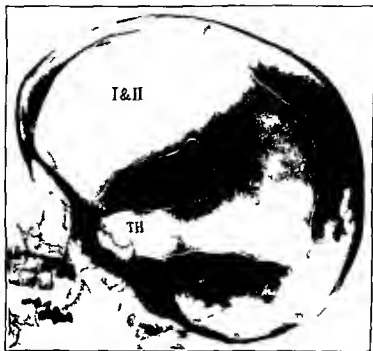


FIG. 969. Lateral and AP cephalograms of an infant a few months old suffering from hydrocephalus. Note the external dilatation of the two lateral ventricles and marked atrophy of the cerebral cortex. TH Temporal horns of the lateral

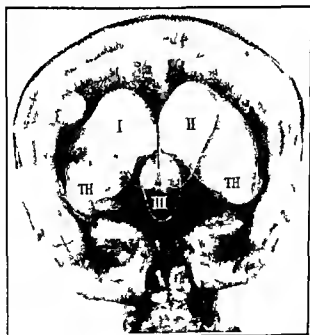
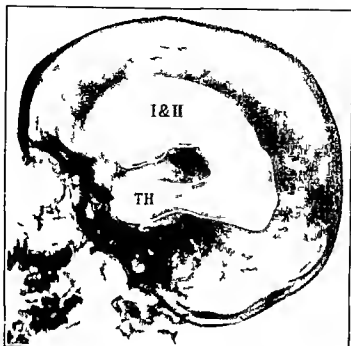


FIG. 263.—Late al and A P encephalograms showing dilated ventricles and moderate cortical atrophy in a patient developing hydrocephalus after the cranial sutures had already ossified. I and II lateral ventricles. TH temporal horns of the lateral ventricles.



whenever signs of increased intracranial pressure appear in a child especially if there are no frank or localizing neurologic signs. Final establishment of the diagnosis in such cases however must frequently await the evidence obtained from air studies which reveal the characteristic dilatation of the ventricles (Figs 261-262-263.)

**Identification of the Type** The differential diagnosis between the obstructive and non-obstructive types of hydrocephalus may now be made clinically with great ease through a simple test devised and standardized by Dandy. This investigator found that a neutral dye injected into one of the lateral ventricles can be recovered by a lumbar tap within twenty minutes in a child suffering from non-obstructive hydrocephalus whereas in the obstructed type forty to sixty minutes or even longer is necessary. Neutral sulphophenol phthalein or neutral indigo-caranine prepared in sterile ampules may be used. In infants with open fontanelles the dye is injected into the ventricles by means of a ventricular puncture performed with an ordinary lumbar puncture needle. In cases where the fontanelles and sutures have already closed trephination is necessary.

If the dye test indicates that the hydrocephalus is of the obstructive type a supplementary test is performed to reveal the site of the obstruction. Spinal fluid is cautiously withdrawn 5 cc at a time and replaced with air or oxygen until 30 to 50 cc of air have been introduced into the spinal canal. After this the child is slowly raised to the full sitting position and roentgen rays are taken of the head. The upper limit of the air as shown in the roentgenograms will indicate the level of the obstruction. The underlying pathologic lesion in any given case can usually be determined only after actual tissue examination.

**Treatment of Hydrocephalus**—The only effective treatment of hydrocephalus is surgical. The first step is to determine the type i. e. obstructive or non-obstructive. This is done by means of the dye test previously described. If this indicates that the hydrocephalus is of the obstructive type the next procedure is to determine the site of the obstruction by means of the air test or modified encephalograms.

**In Obstructive Types**—Treatment should be directed whenever possible to the removal of the obstruction. This is especially true in cases due to expanding lesions such as tumors, cysts or abscesses and also those in which the foramina of Luschka and Magendie have been closed by post-inflammatory cicatrices. In the latter relief can be afforded by making a new opening through the membrane separating the fourth ventricle from the cisterna magna (Fig 264-1 and C).

In cases due to stenosis of the aqueduct of Sylvius a new route

for the escape of the cerebrospinal fluid from the obstructed ventricle is best accomplished by the procedure known as third ventriculostomy. Openings are punched through the anterior wall and floor of the third ventricle in order to allow the diminished up

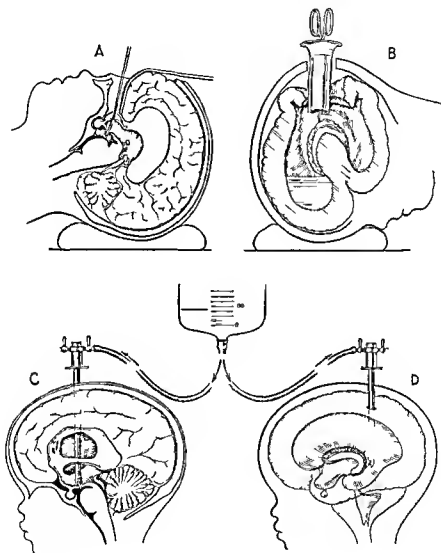


FIG. 264.—A Treatment of obstructive hydrocephalus by puncture of the floor of the third ventricle to permit escape of ventricular fluid into subarachnoid cisterna. B Treatment of non-obstructive hydrocephalus by avulsion of the choroid plexus as originally performed. C New method of treating congenital obstructive hydrocephalus through puncture of floor of the third ventricle by means of a ventriculoscope. D New method of treating non-obstructive hydrocephalus through electrocoagulation of the choroid plexus by means of the ventriculoscope. Note in Methods C and D that collapse of the ventricular wall with attendant shock is prevented by maintaining a constant intra-ventricular pressure.

fluid to escape directly into the large subarachnoid cisterna at the base of the brain. The fluid is thereby drained into the tissue spaces from which it is quickly absorbed by natural physiologic processes. The walls of the third ventricle at these points are extremely thin and the tendency to secondary closure of the openings is very slight.

**In Non-obstructive Types** The treatment of non-obstructive communicating hydrocephalus is directed toward the reestablishment of a normal balance between the rate of production of the cerebrospinal fluid and the rate of its absorption. Since there is no way known to increase the rate of absorption of the fluid from the subarachnoid spaces overlying the cerebral cortex, effort must be directed, as first pointed out by Dandy, toward decreasing the rate of its production through excising portions of the choroid plexuses.

The original technic required complete emptying of the ventricles which allowed collapse of their thin cortical walls. This proved to be too shocking to the patients to make the procedure generally acceptable so that for many years following Dandy's original contribution no further reports of the operation appeared in the literature (Fig 264 B).

A distinct advance in the treatment of hydrocephalus was made with the introduction of the ventricular endo cope or ventriculoscope. This was first used for fulgination of the choroid plexus by Dandy in 1922. Myler (1923) employed it to make an opening through the floor of the third ventricle in a case of obstructive hydrocephalus. Following these early attempts, however, little else was done in this connection until 1934 when Putnam brought out a new endoscope designed especially for electrically cauterizing the choroid plexus and reported a series of cases successfully operated upon. The writer employs a ventriculoscope somewhat different in principle and design which may be used for the treatment of both obstructive and non-obstructive types of hydrocephalus (Fig 264 D).

The results of endoscopic treatment are distinctly promising. Through doing away with the necessity of emptying the fluid out of the ventricles and thus preventing collapse of the ventricular walls, operative shock has been practically eliminated. In a certain number of cases it would appear that the process has been permanently arrested although insufficient time has elapsed for a satisfactory appraisal of ultimate results.

Spontaneous arrestment of hydrocephalus occasionally occurs without intervention. For this reason it is important to be slow in recommending radical measures until there is no doubt but that the hydrocephalus is progressing.

## CHAPTER XLVI

### SPINA BIFIDA MENINGOCELE AND MYELOMENINGOCELE

FAILURE of the dorsal mid line structures to close normally during embryonic life gives rise to three congenital deformities spina bifida meningocele and myelomeningocele (Fig 265) The conditions are closely related to each other and may be regarded as variations of the same basic anomaly In most instances the defect is present at birth or appears shortly thereafter

**Spina Bifida**—The condition is simply an aplasia of the dorsal lamina of the spine and of the spinous processes Most cases occur in the lower lumbar region and as a rule evidence no neurologic symptoms or signs Discovery of the defect is generally accidental following roentgenograms of the area

**Meningocele**—The deformity comprises not only an absence of the dorsal laminae and spinous processes but also a sacculation or herniation of the dura and arachnoid membranes through the bony defect These lesions like spina bifida also occur most often in the lumbar region but may be present at any other point in the spine and even at times through a defect in the occipital bone at the base of the skull

Meningoceles vary greatly in size They may be small flat and fluctuant being loosely covered with normal non adherent layers of skin and subcutaneous tissues or be as wide as the child's back with the overlying skin adherent and so stretched as to be completely translucent Even in extreme cases the communication between the meningocele itself and the meningeal spaces within the spinal canal is always by means of a narrow neck The latter is usually well defined Although it may vary somewhat in size the maximum diameter is limited by the space between the lateral peduncles on each side of the neural canal which in a child rarely exceeds 1.5 cm

The sac has three essential layers the integument dura and arachnoid The latter two layers are almost invariably fused and in large meningoceles all three layers may be inseparable Occasionally the inside of the meningocele is a simple cyst like cavity lined with arachnoid membranes and communicating by a neck with the spinal spaces More often however it is multiloculated in some instances the lobules are freely communicating while in

others there is no apparent intercommunication between them or from them into the spinal spaces. The cavities are generally filled

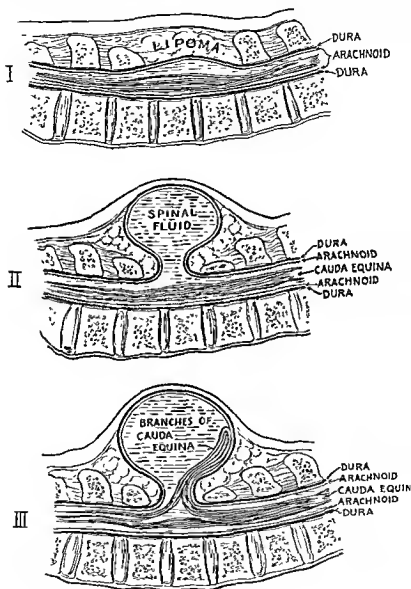


FIG 265 —Diagrammatic representation of: *I*, Spina bifida (occulta) with lipoma. *II*, Meningocele. *III*, Myelomeningocele.

with spinal fluid but may contain mucoid material. In simple types the sac collapses upon being opened; in the multiloculated varieties there may be so much dense stroma between the lobules that even

when the cavities are emptied the meningocele as a whole cannot be effectively collapsed.

**Myelomeningocele** — Spinal nerves, the cruda equina and even the spinal cord itself may herniate out of the neural canal and lie in part within the meningocele. In such cases the condition is termed myelomeningocele. In most instances the nervous tissue elements are firmly adherent to or actually incorporated within the walls of the sac and their dissection therefrom with complete preservation of anatomic continuity and function is often impossible. For this reason the prognosis in myelomeningocele is usually grave.

**Diagnosis** — Simple spina bifida seldom produces symptoms or signs and the diagnosis is based almost always upon accidental roentgen ray findings. No specific treatment is indicated. Meningocele is usually easily recognized. The chief diagnostic feature is the presence of a more or less cystic mass in the dorsal mid line overlying the spine (or mid-occiput) which is firmly attached to these structures. A small multiloculated meningocele with dense stroma may be mistaken for a tumor and a meningocele rising through a low sacral defect may simulate a pilonidal cyst. In either event the proper course is to regard the swelling as a meningocele and advise surgical interference. If at operation the lesion proves to be otherwise it should be treated as indicated.

The differential diagnosis between meningocele and myelomeningocele cannot always be made clinically. When there is paralysis of the extremities or of the sphincters the diagnosis of myelomeningocele is self-evident. There are many cases however without evidence of neurologic signs which at operation reveal nerves so densely incorporated in the walls of the sac that it is impossible to save them. One should therefore offer a most guarded prognosis regarding the function of the lower extremities and sphincters in all cases of apparent meningocele.

**Treatment** — Simple spina bifida requires no treatment. Cases of meningocele should be operated upon at the earliest possible moment since each day's delay increases the risk of pressure necrosis of the skin or traumatic rupture of the sac with consequent meningitis (Fig 266). The common accepted practice is to dissect out the neck of the sac and ligate it and then amputate the sac distal to the ligature. Meningocele of all sizes may be successfully treated in this manner provided there is no infection in the tissues at the time of operation.

**Operative Technic** — The first technical objective is clean dissection and isolation of the neck of the sac at its point of emergence from the neural canal (Fig 267). There is always a well-defined circular or oval foramen with a distinct collar whose diameter is rarely more than  $1\frac{1}{2}$  cm regardless of the size of the sac itself. The

incision is started over the dorsal mid line 1 to 2 inches cephalad to the sac and deepened through the skin and subcutaneous tissues



FIG. 266 —Necrosis of the skin covering a myelomeningocele. Death from meningitis.

until the glistening white surface of the lumbar aponeurosis is reached directly overlying the tips of the spinous processes. This is the proper plane along which to approach the neck of the sac

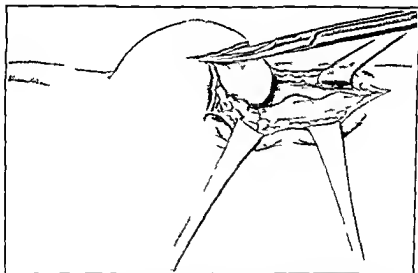


FIG. 267 —Technic for repair of meningocele. Lumbar fascia identified and dissection carried along this plane to the neck of the meningocele.

since the foramen of exit is always through this layer. Once the operator has orientated himself as to depth the incision is carried

toward and onto the sac as far as the skin can be freed from the underlying meningeal layers. From this point the skin and subcutaneous tissue is separated from the rounded dome-like layer of the sac proper with blunt curved scissors. If the sac is a large one every effort should be made to conserve all the skin and subcutaneous tissue possible. (Fig. 268.) The stripping of the skin from the sac is therefore carried as high onto the dome as is possible without cutting into the sac and collapsing it. The lower pole of the incision is treated in the same manner as the upper. The result is an elliptical incision around the sac (a pair of small roughly elliptical incisions with linear extension at each end). The skin and subcutaneous tissues are then retracted to provide exposure

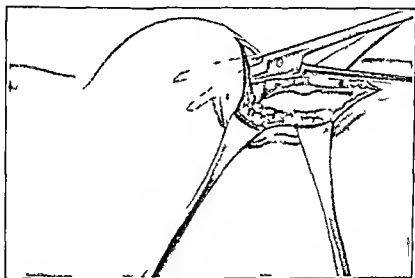


FIG. 268.—The skin enclosing the sac is dissected free and preserved as far as possible to aid final closure.

and the neck of the sac is dissected out cleanly down to the foramen of exit in the fascial layer.

*Opening of the Sac*—The sac should always be opened before the neck is ligated. This is done to permit return of any nervous tissue into the neural canal before the ligation is made. If the meningocele is full and tense it is wise to decompress it slowly through a hypodermic needle before opening it widely, since sudden collapse of the spinal fluid pressure occasionally causes severe shock.

*Treatment of the Sac*—If the inside of the sac consists of a single cavity free from nervous tissue the problem is technically easy. The neck is doubly ligated with silk or chromic catgut and the sac is amputated. However, when the inside of the sac is composed of a number of locules the problem is more difficult. One locule



after another should be carefully opened until the one leading directly into the neural canal is identified. This orientation is important for only in this way is it possible to determine whether or not nervous tissue elements or even the cord itself has entered the meningocele. Any nervous tissue which is seen should be meticulously dissected free of its attachments to the sac wall and be replaced within the neural canal. After this has been done ligation of the neck and amputation of the sac may be safely performed (Fig. 269).

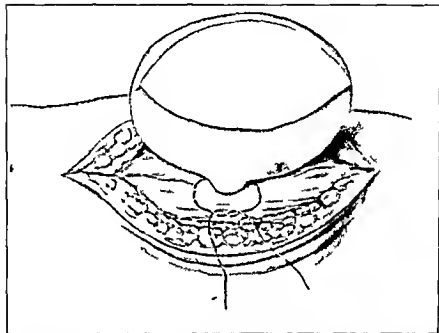


FIG. 69. The neck of the sac is dissected free at its emergence through the laminar fascia and ligated at this point. (The neck is never wider than the neural canal.)

*Closure of the Neck*—Occasionally the neck of the sac has an unusually large diameter or contains so much fibrous tissue in its walls or is so thin that a purse-string suture would easily tear it. In such instances the operator must use his resourcefulness to effect the closure. This can always be done by pinching the walls of the cyst just distal to the neck and suturing the layers in place with fine interrupted silk sutures. The closure of the neck should always be reinforced by overlapping adjacent layers of the deep fascia (Fig. 270).

*Closure of the Skin by Pedicle Graft*—Closure of the skin defect after excision of a large meningocele may prove to be a problem

in plastic surgery. Even after all possible skin has been saved by making the initial incision in the manner described and maximum undercutting and sliding of the skin has been performed the skin

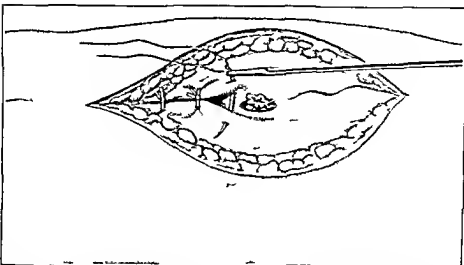


FIG. 270—Closure of the hatus in the lumina face a by plication of adjacent tissue. Skin defects are closed in appropriate manner using a pedicle flap if necessary.

edges may still fail to meet. Moreover, the defect is usually directly over the stump of the sac. In such instances it is necessary to slide a full thickness pedicle graft from either the back or flanks (Fig.



FIG. 271—Subcutaneous meningocele after treatment.

273) It is important therefore to sterilize as great an area of skin over the back and flanks as may be required. The pedicle flap must be large enough to cover the stump generously and to lie in place

without tension on its suture lines. The area from which the pedicle graft has been taken is covered with pinch grafts. These can generally be taken from the thin layer of epithelium attached to the dome of the excised sac. Strict asepsis is necessary throughout all stages of the operation since the slightest contamination is inevitably communicated to the spinal meninges and results in fatal meningitis.



Fig. 272



Fig. 273

Figs. 272 and 273. Meningocele before and after treatment. Skin closure by pedicle flap.

**Relationship of Meningocele to Hydrocephalus**—The relationship of meningocele to hydrocephalus is controversial. It is unquestionably true that both conditions occur frequently in the same patient. They may develop concurrently or the hydrocephalus

may only begin to be noticed after the meningocele has been operated upon. When this occurs the claim is often made that the hydrocephalus is the result of the removal of the meningocele. It is stated by some authorities that the tissues contained within the meningocele actively assist in some manner in the absorption of cerebrospinal fluid and that the removal of the meningocele reduces the rate of absorption sufficiently to produce a communicating type of hydrocephalus. Penfield argues against ligation and removal of the sac and urges that the sac be opened, evacuated of its contents and then plicated and sutured in folds to form a flat pad overlying the foramen of exit of the meningocele.

There are many arguments contradicting this viewpoint and opposing this type of treatment. The chief fact would appear to be that the two conditions occur simultaneously so often in the newborn that it seems proper to regard them simply as different manifestations of a common developmental deficiency. Nor is it significant that in many instances hydrocephalus is only observed after a meningocele has been excised. Meningoceles are commonly well developed at birth, whereas hydrocephalus usually requires weeks or even months to declare itself with sufficient certainty to justify the diagnosis. Moreover the commonly accepted practice of treating meningocele by simple ligation and excision of the sac has given excellent results in the hands of many observers.

The theory that the spinal arachnoid plays an important role in the absorption of cerebrospinal fluid has never been satisfactorily established. Attempts to produce hydrocephalus experimentally in animals by isolating the spinal arachnoid from the cranial arachnoid system have been unsuccessful. Moreover complete blocks in high thoracic levels are frequent in cases of severe spinal injury yet the patients never develop increased intracranial pressure. Finally the tissues of which the meningocele is composed are frequently unsuited anatomically both in gross and microscopic structure for any such specific and important function as the absorption of spinal fluid. Even in cases where the tissues making up the sac might be compatible with such a function the actual size of the sac is so small in comparison with the entire pia arachnoid system of the brain and cord that it seems incredible it could possess such a determinate role in the development of increased cerebrospinal pressure.

Finally, plication of the sac, as advocated by those who believe that hydrocephalus is a result of excision of the sac, is in the writer's experience only occasionally possible. At least one-half of the meningoceles are multilocular and have interocular septa of such thickness and firmness as to preclude effectively any attempt at plication.

Meningoceles of the occipital region are treated according to the same principles as meningoceles of the spine.

**Cases Suitable for Operation.**—The selection is a matter for considerable thought. In this connection it should be clearly understood that no anatomic or physiologic defect in nerve structure or function is ever improved by operation. The latter removes an unsightly mass and prevents the development of a spinal fluid fistula with resulting meningitis. It is an act of very doubtful charity to repair a myelomeningocele in a child who is already paralyzed in its legs and sphincters. Although often life-saving, it at the same time dooms the child to an existence of the most pitiful invalidism.

It is the writer's practice to refuse to operate upon a child with meningocele when the legs and sphincters are paralyzed. Involvement of the legs is easily determined by pricking the soles of the feet with a pin. A normal child cries with pain and quickly withdraws its foot, thereby demonstrating normal sensory and motor functions. Paralysis of the sphincters is determined by rectal examination. Normally, the sphincter is tight and resists the entrance of even the tip of the little finger. A forced entrance is painful and causes the child to cry. The absence of such sensory and motor reflexes indicates paralysis.

## CHAPTER XLVII

### EPILEPSY

EPILEPSY is a symptom of disease rather than a disease itself. An epileptic fit indicates a brain which is either pathologically irritated or pathologically irritable. The primary etiologic factors capable of setting up either of these processes are numerous, differ widely in character and may be specific or non specific.

**Etiology—A Localized Pathologic Lesions** of a gross anatomic nature are a common cause of epilepsy. In this group of etiologic factors are included such lesions as congenital arachnoid (porencephalic) cysts, gummata, tuberculomata, post-traumatic scar tissue formation, abscesses of the brain and intracranial tumors (Figs 274-275-276). These and other lesions are capable of powerfully irritating the brain. Since the irritation produced by such lesions is fairly well localized, the clinical reaction tends also in whole or in part to be focal in character. This is the basis of focal epilepsy, which will be discussed more fully later.

In addition to these gross anatomic lesions there are also other localized lesions which are not directly visible to the eye and which may not be actually anatomic in character but which act in the same way as anatomic lesions in producing focal epilepsy. These consist essentially of sharply localized areas of hyperirritability which act as trigger zones for setting off epileptic fits. They can only be discovered by electric stimulation of the cerebral cortex but their ablation often relieves the patient of attacks (Fig 277).

**B Diffuse Pathologic Processes** essentially physiologic rather than anatomic in character may also cause epileptic attacks, as is witnessed by the fact that the most exhaustive study of the brains of many epileptics reveals no significant anatomic lesion. *Some congenital developmental defect* which leaves the cortex of the brain more sensitive to ordinary stimuli than is normal is most probably the primary factor in all these cases. Also study has revealed that various secondary mechanisms appear able to effect this threshold favorably or unfavorably and have a secondary role in the release of convulsive phenomena.

*Different types of secondary release mechanisms* may operate in different cases of epilepsy. For instance, in the convulsions which frequently usher in acute febrile illness in children, the release mechanism might be either the toxins elaborated by the invading

bacteria or the *hyperpyrexia*. The convulsions of *uremic poisoning* are undoubtedly released by the highly concentrated nitrogenous products of metabolism circulating in the blood stream which would

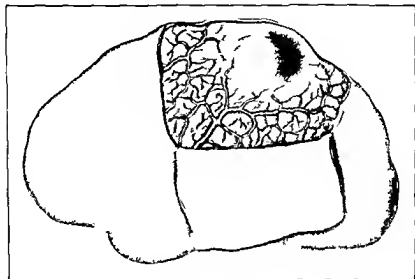


FIG 274 —Cyst or tumor The former may either be congenital or neoplastic in origin

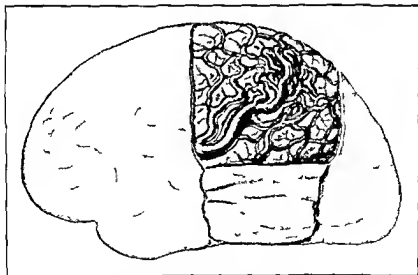


FIG 275 —Congenital vascular anomaly

normally have been eliminated by the kidneys. The convulsions of *eclampsia* are due to a still different release mechanism. It has been

found that an unfavorable *water balance* or *acid base balance* within the body tissues sometimes effects the incidence and severity of convulsions in epileptics, this forms the basis on the one hand for

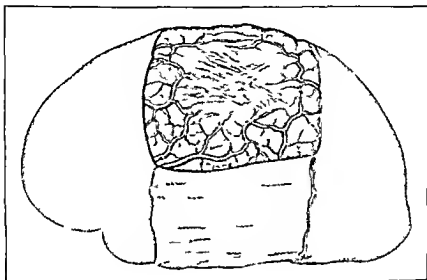


FIG 276 —Cortical retraction (post traumatic)

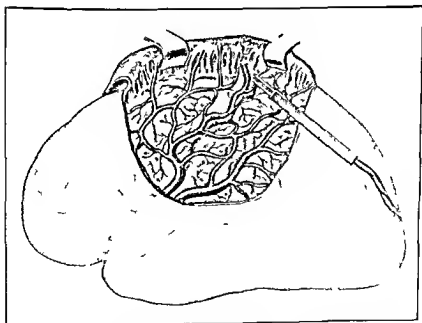


FIG 277 —Epileptogenic zone without morphologic lesions identified only by electrical stimulation of the cortex



the "dehydration" therapy, and for the "starvation" treatment and "high-fat" (or ketogenic) diets on the other. We know, too, that hypoglycemia (hyperinsulinism) occasionally induces epileptiform seizures.

*Other biochemical mechanisms* may also influence the incidence of convulsions. Since the *Na-ion concentration* in the body fluids has been shown to be a marked secondary factor in producing the violent irritation of the inner ear characteristic of Ménière's syndrome, it might well have a similar rôle in regard to other irritative processes affecting the central nervous system. The effects of *dietary and vitamin deficiencies* upon the nervous system have been indicated anew in recent studies which suggest a possible rôle for these factors in the mechanism of epilepsy. Finally, the relationship between *allergy* and epilepsy is a subject which merits further study.

Epilepsies with this type of etiology fall naturally within the domain of the biophysicist and biochemist rather than the pathologist, and must be treated by the medical therapist rather than the surgeon. Unfortunately, the problems of etiology and therapy to be solved in these cases are more complicated and obscure than those presented by gross anatomic lesions, and as a matter of fact are often very little or not at all understood at present. These types of epilepsy, usually congenital or beginning in early childhood, and without focal clinical features or demonstrable pathologic processes in the brain to explain the attacks, are designated "*idiopathic*" epilepsies.

The term "idiopathic" epilepsy should, however, be used reluctantly, and with the thought that it refers not so much to etiology undeterminable as to etiology not yet determined. At present there is about a real renaissance of interest in the etiology and mechanisms of the epilepsies and it seems probable that as study continues the great heterogeneous category of "idiopathic epilepsy" will be slowly fragmented into smaller subgroups which will have more sharply defined clinical syndromes based on constant pathologic factors which can be determined and successfully treated by specific therapy.

**Clinical Forms** —All cases of epilepsy may, from a clinical standpoint, be classified as (1) focal or (2) non-focal.

**Focal Epilepsy** —Focal epilepsy, broadly speaking, is any epilepsy caused by a specific localized irritating lesion on the cortex of the brain. This type may be treated surgically and offers a favorable prognosis.

**Non-focal Epilepsy** —Non-focal epilepsy, on the other hand, is the result of one of the diffuse, non-specific pathologic processes above described, often more physiologic than anatomic, frequently not understood, and hence termed "idiopathic." As pointed out, these cases fall within the field of the medical therapist, with

the prognosis at present, due to the complexity of the problem, far less favorable than that for focal epilepsy.

Because the prognosis for focal epilepsy is so far more favorable than for the non-focal type, every resource available to the clinician should be utilized to demonstrate if possible a focal origin for all epileptic patients.

*Focal convulsions* are indisputable evidence of a discrete, localized irritative lesion of the brain. The classical example is the so called "Jacksonian" fit. Here, for instance, clonic movements first appear in the digits of one hand, pass slowly up the arm involving progressively the wrist, elbow, shoulder, face, trunk and leg of the same side, finally involving the entire body in a generalized clonic convulsion accompanied by loss of consciousness and incontinence of the sphincters. With such a convulsion the diagnosis of focal epilepsy, due to a focal lesion, is easily established.

*General convulsions*, however, do not rule out focal epilepsy. Indeed, generalized convulsions are very frequently produced by the most sharply localized lesions, as, for example, brain tumors. Particularly is this true in the later stages of the tumor growth when the entire brain has been indirectly involved and is abnormally irritable. A person observing only a convulsive seizure in such a patient might see no evidence whatever from the character of the attack itself that it was produced by a localized lesion which was capable of surgical removal.

All the available sources of focal data should be painstakingly examined in every case of epilepsy. Localizing features of great importance, not at first apparent, may many times be brought to light thereby. The possible sources of this information are herewith enumerated and discussed.

1 *The early convulsion* occurring in the initial stages of a long-standing case of epilepsy, which now shows only generalized major convulsions, is sometimes found upon careful questioning to have been definitely focal in character.

2 *Abortive or incomplete attacks*, in which only the early part of the convulsive pattern is acted out, and the "fit" is arrested spontaneously before becoming generalized, often yield valuable localizing information which is altogether lacking in the more serious generalized attacks. For instance, a patient who suffers currently with two or three attacks of generalized convulsions a month may experience from time to time between these attacks a transient stiffening of the fingers of one hand or feeling of heaviness in one leg, or a difficulty in expressing himself as fluently as usual.

3 *The aura or warnings* which often immediately precede generalized convulsions are occasionally of value in establishing the focal nature of epilepsy, especially if the auras present a pattern which is constant in recurrence. For example, bright lights referred

constantly to the right hand fields of vision of both eyes just before a convulsion, might well indicate an irritative lesion in the left temporal or occipital lobe

4 *The initiating phase* in any given attack may also give leads which will help to lateralize or even localize a lesion. Sometimes this will be purely sensory such as a feeling of thickness on one side of the tongue or a warm feeling passing down an arm or a heavy feeling in the leg followed almost immediately by loss of consciousness and a bilateral non focalizing convulsion. Occasionally the very first movement in a truly generalized convulsion may repeatedly and consistently be a stiffening of one leg or a raising of one arm or a turning of the head always to one side or a twitching of one side of the face. These may occur just as the patient is losing consciousness or even after he is unconscious and may precede the general fit by only a fleeting second. They could not properly be called Jacksonian phenomena but if they constantly initiate the convulsive pattern they probably have definite relationship to a trigger area in the brain. Even the simple observation that convulsions begin always on one side of the body may be of great value in locating a lesion.

5 *Postconvulsive sequelæ or residual symptoms and signs* remain even a short while after the attack itself has passed may give true focal data. For example. Following complete return of consciousness a patient may for several hours or days drag one foot in walking or have a conspicuous difficulty in speaking although mentally quite clear.

6 *Other symptoms or signs referable to the central nervous system* found in association with convulsions should automatically raise suspicion of a focal lesion. Headache nausea blurring vision weakness or numbness of an extremity or changes of personality exhibited in a patient having epileptic attacks would constitute strong presumptive evidence that an expanding lesion such as tumor or abscess was producing the seizures. All effort should be made to establish the existence of such a lesion if necessary by air studies.

7 *Ictive pathologic processes* elsewhere in the body such as pulmonary abscess tuberculosis syphilis or any neoplastic disease in a patient suffering with convulsions should always raise suspicion of a metastatic lesion of the same type in the brain.

8 *Trauma preceding the onset of epilepsy* offers presumptive evidence of localized scar tissue or adhesions of the brain even though the convulsions themselves are generalized and without focal features. This is so for the reason that the scar tissue may be present in silent areas such as the frontal lobe taken up only with the higher psychic functions from which sharply focalized motor or sensory response is impossible.

9 *Röntgen ray examination* often establishes the presence of a focal lesion by depressed fractures or by revealing erosions, hyperostoses, vascular anomalies of the bone or even calcific deposits within the brain itself which indicate the presence of a tumor.

10 *A study of epilepsy is rarely complete without an encephalogram or ventriculogram.* These are special forms of roentgen ray study in which the spinal fluid is removed from the ventricles and subarachnoid spaces of the brain and replaced by air or other gas which shows in great contrast in roentgen ray pictures. These frequently reveal distended or distorted ventricles or abnormalities of the subarachnoid spaces which undeniably establish the site and nature of the pathologic process accountable for the convulsions when all other methods of study have failed. For example an encephalogram or ventriculogram may be the only way possible to discover and accurately locate a congenital porencephalic cyst in an infant having convulsions which would otherwise be regarded as a case of congenital idiopathic epilepsy.

11 *Cerebral exploration and cortical stimulation in search for a trigger zone* may be carried out in selected cases when all other attempts to establish a focal etiology have failed. This technic occasionally discloses focal features not otherwise possible of detection. Although this approach to the problem of epilepsy is not new in principle its general and systematic use in the study of epilepsy has only recently been undertaken in a few of the neurosurgical clinics. The method consists essentially of exposing under novocain anesthesia the cerebral hemisphere under most suspicion. In a systematic way stimulation of the cortex is carried out using a fine bipolar electrode and a minimal current—galvanic or faradic. As the investigator stimulates point after point along the various gyri sharply focalized responses such as the movement of a single finger or tip are normally obtained. But if a spot is found which is a so-called trigger zone for convulsions its stimulation even with a very weak current causes a violent convulsion and in typical cases reproduces the exact pattern of the epileptic fit from which the patient seeks relief. Excision of this trigger zone will in favorable cases terminate the attacks.

By means of electrical stimulation the writer discovered in certain patients that areas of the brain to which pachionian granulations were attached appeared to be more sensitive than the cortex elsewhere and were virtually trigger zones. It would appear therefore that in these particular cases the granulations acted like adhesions. By dividing the granulations and freeing the brain from its rigid attachment to the dura at these points the incidence and severity of the convulsions were greatly lessened. This example is cited to show that with more exhaustive search new types of focal epilepsy are being discovered.

The diagnosis of idiopathic epilepsy is permissible only when (1) the disease first appears during infancy or early childhood and (2) when no evidence of a specific or localized etiology can be established by any of the methods of study outlined above. Even then the diagnosis should be considered tentative. It should be made with hesitation and with the same amount of mental reservation as would accompany a diagnosis of fever—unexplained. For it is unquestionably true that as a result of improved methods of study and a revival of interest in the subject many cases of epilepsy which would have been regarded as idiopathic (and hence incurable) twenty years ago are today being shown to be the result of a localized specific lesion for which surgery offers a favorable prognosis.

The term *grand mal* has been applied in the past to any major epileptic attack characterized by sudden loss of consciousness, clonic convulsions of all extremities, biting of the cheeks and tongue and incontinence of urine or feces.

The term *petit mal* designates only a momentary loss or alteration of consciousness without convulsive movements. This may be only a very early and immature phase of a convulsive pattern which will later on evolve into a true major epilepsy or it may be the only ultimate which the convulsion will assume. In children it practically never represents the final form of the epileptic pattern; in adults past middle life, especially those with cerebral arteriosclerosis, it is often the final form. Here the spell may possibly be on a vascular basis representing a sort of intermittent claudication of the brain.

**Treatment of Epilepsy**—The first step in treatment is to determine whether it is focal specific epilepsy and hence surgical or non focal non specific idiopathic epilepsy and therefore a medical problem. Every resource available as outlined above should be utilized for this purpose since the outlook is so much better if a focal cause for the attack can be found.

**Surgical Therapy**—Epilepsy due to a specific localized lesion of the brain should be treated by direct surgical attack upon the lesion designed to correct or irradiate it. Depressed fractures should be elevated and splinters of bone removed, adhesions should be freed and scar tissues excised, cysts or abscesses drained, tumors removed and cortical trigger zones ablated. The correct procedure will in each instance be determined by circumstance and the surgeon's judgment. The prognosis will depend upon the nature of the lesion and the operator's skill but in general should be favorable.

**Non surgical Therapy**—Epilepsy with an undetermined non specific non focalized etiology so-called idiopathic epilepsy must be treated in a quite different manner. In these cases the trial and error method must be employed. All the various theories

regarding the etiology and treatment of idiopathic epilepsy must be considered. The ketogenic the high protein the low salt and low fluid diets should be given a thorough trial. The possibility of hypoglycemia should be ruled out. One or the other may be found empirically to be of definite value. Allergy must also be studied as a possible factor and vitamin deficiencies excluded as contributory causes.

*Luminal* affords excellent palliative therapy in the purely symptomatic management of epilepsy particularly the idiopathic type. Its action is entirely that of a sedative but it does unquestionably reduce the number of attacks has practically no ill-effects and can be administered indefinitely without addiction or development of tolerance. An average dose for an adult would be 1 grain t i d for a child proportionately less perhaps  $\frac{1}{2}$  to  $\frac{1}{4}$  grain t i d.

*Bromides* also reduce the frequency and severity of convulsions but have one drawback in that prolonged usage frequently causes eruption of the skin. They are chiefly employed as temporary substitutes for luminal in long standing cases.

The management of a person during an epileptic fit deserves brief comment. The chief concern is to prevent the patient from injuring himself. As soon as an attack begins he should be placed immediately on the floor so that he cannot hurt himself by falling. A wedge preferably of wood should then be forced in between the upper and lower teeth to prevent the tongue being bitten. It is important to insert the wedge between the molar teeth and not the incisors since the latter may be broken off. A good wedge can be made from a clothes-pin split longitudinally. A piece of wood with a blunt end is worthless since it cannot be introduced between the closely clenched teeth.

Once the teeth are blocked the patient should be rolled well over on his side or face with the forehead resting on the extended arm. This allows mucus to flow out of the mouth instead of being aspirated into the bronchi and lungs. It also permits the tongue to fall forward clearing the glottis and reducing the degree of mechanical asphyxiation and cyanosis which is such a distressing feature of major convulsions. There is little else to do except to wait for the attack to pass.

**The Cerebellar Fit**—The so-called cerebellar fit merits discussion since its differential diagnosis from the epileptic type is occasionally necessary and extremely important.

The cerebellar fit is in reality a transient episode of decerebrate rigidity. This is brought about in most instances when pressure of an intracranial tumor usually cerebellar pushes the brain stem downward against the edge of the foramen magnum so sharply that the pressure of the edge interrupts the functional continuity of the major tracts leading from the spinal cord to the cerebrum. When

this occurs the patient goes into a state of acute opisthotonus with spastic hyperextension of all extremities. Although there may be fine tremors there are no convulsive movements. All the extremities are involved at the same time and to the same degree. Consciousness may or may not be lost.

Although attacks of this sort usually indicate the presence of a cerebellar tumor they may also occur with any acute obstructive hydrocephalus secondary to brain tumor. The situation is usually brought on by straining often while the patient is on a bed pan. Sharp sudden antiflexion of the head or neck will also at times initiate an attack in a patient who has an advanced cerebellar tumor.

Cerebellar fits are rather frequent in children since the most common intracranial tumor of childhood is the mid line cerebellar tumor. The attacks are prone to occur with *antiflexion of the head and neck* as this seems to pull the brain stem down through the foramen magnum. An enema is also a frequent provocative. This may well be the result of the incidental antifixion of the head and neck caused by the elevation of the buttocks and pelvis onto the pan or may be due to the increased intracranial pressure which occurs with straining. Very sick children with cerebellar tumors are not given regular enemata or permitted to use a bed pan. Rather rectal instillations of 25 per cent aqueous solution of magnesium sulphate (25 to 100 cc) are administered and a sort of diaper applied which contains absorbent material. Such a child is better off sleeping on its side or if on its back without a pillow.

**Treatment** — This comprises the operative relief of the medullary pressure taking place at the foramen magnum and at the same time if possible removal of the lesion producing the pressure or the hydrocephalus. These measures should only be attempted by the trained neurosurgeon.

## CHAPTER XLVIII

### INTRACRANIAL TUMORS OF CHILDHOOD

INTRACRANIAL tumors occur far more frequently in children than is commonly supposed. Their two sites of election are (1) the mid line of the cerebellum and (2) the region of the pituitary body.

#### CEREBELLAR TUMORS

Cerebellar tumors are the most common intracranial growths in early life. They arise chiefly from the mid line or vermis and are of two types the medulloblastoma and the astrocytoma named after the dominant cells found in each.

**Medulloblastoma**—This is the most common intracranial tumor of early life and occurs only during childhood. It arises almost without exception from the cerebellar vermis or mid line and from the roof of the fourth ventricle and the clinical symptoms and signs which it produces are the natural result of its position. It grows rapidly and as it expands the tumor presses downward and tends to obliterate the fourth ventricle (Fig 257). This interferes with the free circulation of the spinal fluid at this point and produces hydrocephalus with all its accompanying clinical manifestations. As the growth spreads laterally and invades the two cerebellar hemispheres it produces typical cerebellar ataxia in the arms and legs. It should be noted however that since the tumor develops from the roof of the fourth ventricle it rarely produces pressure effects on the cranial nerves as these arise from nuclei which are located in the medulla oblongata or floor of the fourth ventricle.

The tumor is non encapsulated and highly invasive. In the gross it is so cellular and soft that it is best removed by suction. Microscopically the growth has a superficial resemblance to lymphoma in that it consists of small round homogeneous cells closely packed without special pattern. Transplantation to the spinal cord may occur by way of the spinal fluid but metastasis through the blood stream is unknown.

**Symptomatology**—The dominant clinical features consist of (1) evidences of increased intracranial pressure and (2) ataxia. In certain instances signs which strongly suggest meningeal irritation may also result. A typical case may run a course as follows. A child two or more years of age who has previously been well and active begins to get unsteady on its feet. At first this



does the rapidity of onset and course of the illness exclude tumor since in many cases only three or four weeks elapse between the appearance of the first symptom of a particularly malignant medulloblastoma and the terminal stage of the disease. The history is frequently of great value especially if the illness began with an insidious and slowly progressive disturbance of gait which preceded by a substantial length of time the appearance of the acute intracranial symptoms. Papilledema of the optic nerve head or choked disc is of prime importance and is strong evidence in favor of tumor. Although the existence of fever and leukocytosis suggests infection it must be remembered that dehydration due to persistent vomiting may also produce both of these.

At times acute meningitis can only be excluded through examination of the spinal fluid. Chronic tuberculous meningitis may also be difficult to differentiate. The long history which often accompanies the latter infection may suggest a neoplasm. The history of exposure and the existence of foci in other parts of the body of course are important. The Mantoux test for active tuberculosis should also be utilized. But frequently as in the case of acute pyogenic meningitis final differentiation must await examination of the spinal fluid.

*Tapping the spine* in cases of doubtful diagnosis should be performed with great care since the sudden withdrawal of only a few cubic centimeters of fluid from the spinal spaces may allow the medulla oblongata to prolapse into the foramen magnum and produce sufficient pressure against the respiratory centers to cause death. The tap in suspected cases should therefore be done with the patient in the horizontal position using a fine needle and removing the fluid a drop at a time and taking only sufficient fluid to permit of immediate cell count. If the fluid is clear and the cell count normal the needle should be immediately withdrawn without further removal of fluid. Xanthochromia is suggestive although not definitely diagnostic of tumor.

*Ventriculography*—This procedure is occasionally required to establish the diagnosis of cerebellar tumor. When air studies are necessary they should always be carried out by trephination and the direct introduction of air into the ventricles. The method of performing air studies by the introduction of air into the lumbar arachnoid spaces is definitely dangerous in the presence of cerebellar tumors for the same reason as diagnostic spinal puncture. Cerebellar tumors typically produce interference with the passage of cerebrospinal fluid through the fourth ventricle and thus cause dilatation of the third ventricle and the two lateral ventricles. When this condition is revealed in the ventriculograms it gives strong confirmation to the clinical diagnosis of cerebellar tumor.

**Treatment.**—This consists of the surgical exposure of the medulloblastoma and its attempted eradication. The prognosis, however, is poor since complete removal of this particular type of tumor is practically always impossible, even after the most radical surgery and supplementary roentgen-ray therapy recurrence is the rule. If it were possible to be certain without exploration that the lesion of the cerebellum was in reality a medulloblastoma, surgical interference would hardly ever be justified. Unfortunately, however, it is not possible clinically to differentiate this tumor from other cerebellar lesions which offer a more favorable prognosis. For this reason, and until the clinical methods of diagnosis have been improved, exploratory operation is necessary.

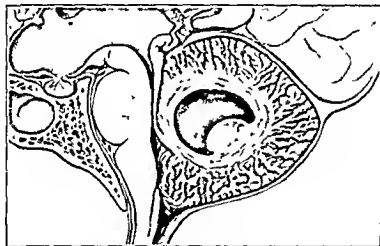


FIG. 278 Astrocytoma compressing the floor of the fourth ventricle and medulla causing obstruction

**Astrocytoma.**—Like medulloblastomata, the astrocytomata also arise from the cerebellar vermis. The tumor receives its name from the fact that its chief cellular component is the "astrocyte" or star-shaped cell. This cell sends out multiple slender fibrils in various directions which interlace with similar fibrils of other cells to form the finer structural framework of the brain. Because they are highly mature with very specialized functions, the cells multiply slowly. As a result, astrocytomata are relatively benign tumors. They are peculiar in their frequent tendency to the formation of cysts (Fig. 278). In such cases the active tumor tissue is usually confined to a single nodule protruding into the cystic cavity from its walls. Removal of the nodule alone, without removal of the entire cyst wall, is sufficient to effect a permanent cure.

**Symptomatology**—The clinical picture produced by cerebellar astrocytoma is very similar to that of medulloblastoma except the symptoms and signs develop much more slowly. Differential diagnosis has been considered under the discussion of Medulloblastoma.

**Treatment**—This calls for exploratory operation incision and drainage of the cyst and extirpation of the tumor nodule arising from the cyst wall. Whenever this is possible a permanent cure may be expected. Even if total extirpation of the tumor is impossible the prognosis is not altogether bad as the growth rate is exceedingly slow. Cases are known to be well many years after removal of the tumor or even simple evacuation of the cyst.

### TUMORS OF THE PITUITARY ANLAGE

Congenital tumors and cysts arising from the pituitary anlage comprise the second most important group of intracranial neoplasms occurring in children.

**Pathology**—The pituitary body is formed by the fusion of a downward evagination from the floor of the third ventricle with an upward evagination from the buccal mucosa, the so-called cranial buccal pouch. These form the anterior and posterior lobes of the pituitary body respectively. Anomalies in the involution of the cranial buccal process often leave cell rests in the vicinity of the anterior lobe and at times even a portion of the epithelial lined cranio bucco tube may remain as a small cyst within the sella turcica. These vestigial remnants may later resume growth and give origin to various types of congenital tumors and cysts in the immediate vicinity of the pituitary body. By far the most important of these is known variously as cranio-buccal pouch cyst, Rathke's pouch cyst or suprasellar cyst (Fig 200). For purpose of discussion the first of these terms will be used.

**Clinical Manifestations**—A cranial buccal cyst usually starts to grow within the sella turcica itself. In this location it first disturbs or destroys by pressure the active endocrine cells of the pituitary gland thus affecting skeletal growth, sexual development and fat metabolism. The well known Frolich syndrome is frequently a result. As the cyst continues to expand the sella itself becomes enlarged. As this process continues the membranous diaphragm which closes over the top of the sella turcica is stretched upward and pressure is brought directly against the under surface of the optic chiasm. This produces a progressive primary optic atrophy with quadrant or heminoptic (bitemporal) defects in the visual fields. Eventually the diaphragm selle may rupture allowing the cyst to escape through the tear so that a part of the cyst comes to rest above the sella within the cranial cavity proper. The

formation resembles a collar-button abscess. Continued growth of the cyst in this location produces, in addition to the original pituitary syndrome, symptoms and signs due to pressure upon the cerebral structures which are similar to those produced by any other intracranial neoplasm. Signs of increased intracranial pressure may thus result, or acute hydrocephalus develop from pressure of the cyst upon the two centrally placed foramina of Monro.

The cysts may develop very slowly or they may be present a long time before producing symptoms. Under such conditions deposits of calcium frequently form in their walls (about 80 per cent of the cases), such findings are of considerable value in confirming the clinical diagnosis through roentgen-ray studies.

**Diagnosis.**—The essential clinical finding is the presence of a more or less typical pituitary syndrome associated (in 80 per cent of the cases) with calcification within or above the sella turcica. It is not always possible, especially in very young or uncooperative children, to be sure of the type of "eye signs," particularly defects in the visual fields. When, however, blindness begins insidiously and progresses actively and is accompanied by an increasing optic atrophy in a child who previously has had good vision, and when no other definite pathologic cause for the blindness can be found, the possibility of a suprasellar cyst must always be considered. Even though a positive diagnosis cannot be made, exploration of the sella, in the writer's opinion, is justified.

**Treatment.**—This consists of the surgical evacuation of the cyst. The procedure requires the skill of a trained neurosurgeon, and in competent hands entails only secondary risks. When performed in time, the operation affords immediate and substantial return of vision. In a certain proportion of cases, however, the cysts refill and reproduce their previous pressure effects. The ultimate prognosis should therefore be guarded.

### CEREBRAL TUMORS.

Cerebral tumors are so very rare in children that their consideration will be omitted in this limited discussion of intracranial neoplasms.

## CHAPTER XLIX

### BRAIN ABSCESS

APPROXIMATELY 83 per cent of intracranial abscesses originate from chronic suppuration of the ear and 9 per cent from infection of the nasal cavity and accessory sinuses. In view of the great frequency of otitis media and mastoiditis in children the importance of these conditions as etiologic factors in the production of cerebral abscesses cannot be too strongly emphasized. Moreover chronic otitis media frequently produces mastoiditis which especially in infants is difficult to diagnose. The following is a good rule to observe. Whenever headache, nausea, vomiting, visual disturbances or stupor appear in a child who has had a chronically discharging ear, especially if the ear has recently and spontaneously stopped draining, abscess of the brain should be strongly suspected. The same principle applies to cases in which symptoms of intracranial pressure develop following chronic suppuration of the frontal or other accessory nasal sinuses.

**Pathology**—Although the usual site for cerebral abscess of otitic origin is in the temporal lobe just above the petrous ridge, the process may spread so as to involve a great part of the lobe. In most instances the abscess centers at a point about 1 inch above and behind the external auditory meatus. Such infections occur practically always by direct invasion through the dura from necrotic mastoid cells. Suppuration apparently secondary to chronic otitic infections may occasionally develop in other parts of the brain such as the frontal lobe and even in the opposite hemisphere. While this fact is well recognized the method by which distant parts are infected is not understood. An abscess arising from the accessory nasal sinuses usually develops in the frontal lobe on the same side as the diseased sinus. Metastatic abscess, most often from the lung, may occur in any part of the brain but is most frequently encountered well up on the cerebral hemisphere anterior to the motor zone.

The cellular pathology of brain abscess is similar to that of an abscess in any other part of the body. Immediately after the introduction of infection into the brain tissue there is a diffuse cellulitis (or encephalitis).liquefaction gradually takes place in the center of the inflammatory zone and a walling-off reaction develops about the periphery. In favorable cases this latter process progresses

until a firm wall is formed from connective tissue and glomatous elements. In chronic cases this may be 3 to 4 mm. in thickness and be quite tough and the infective organisms seem to progressively lose their bacteriologic virulence. Usually however the abscess wall yields and the purulent collection expands in size. If its growth be slow and the reaction of the surrounding tissue be slight the abscess may eventually fill the entire temporal or frontal lobe with amazingly few clinical symptoms or signs. Before reaching such size however capsular rupture generally occurs and the pus drains into one of the ventricles or into the subarachnoid spaces. In such cases meningitis invariably follows with fatal outcome.

**Symptomatology**—The clinical symptomatology of cerebral abscess merits special comment. In some respects the process tends to produce the same symptoms as a tumor occurring in a similar location. For instance in addition to signs of general increased intracranial pressure an abscess of the temporal lobe usually produces an homonymous hemianopsia in the visual fields of the opposite side and if the process is on the dominant side of the brain some type of aphasia also results. An abscess of a frontal lobe tends to produce changes in personality. However the extent of these changes varies amazingly and bears no definite relationship to the size of the abscess itself. Small abscesses especially when acute and associated with edema in the surrounding tissues may produce gross clinical signs whereas larger older and more encapsulated types frequently produce a minimum of signs and certainly far less than a tumor of equal size. Furthermore a definite encapsulated process may produce very few local signs. This is probably due to the fact that the abscess distributes its pressure effect more uniformly over greater surfaces than a firm tumor so that its mass action is translated more into general pressure than into focal pressure effects.

The constitutional reaction to the infection may also be extremely mild or entirely lacking even with an abscess of considerable size. The pyrexia rarely exceeds a degree or two while the leukocytic count usually approximates 12,000 to 15,000.

**Diagnosis**—At times it is very difficult to establish the diagnosis and localization of cerebral abscess from purely clinical evidence when the signs of intracranial pressure or irritation develop during the course of chronic mastoiditis or suppuration of an accessory nasal sinus. Although the correct presumptive diagnosis should be cerebral abscess its actual confirmation must usually await operation. Localization of the abscess is determined partly on the law of averages and when these fail by certain diagnostic operative procedures. Cerebral abscesses originating from the ear occur in the temporal lobe of the same side and are centered most often

beneath a point approximately 1 inch above and 1 inch behind the external auditory meatus at a depth of 1 to 3 cm. In doubtful cases a small trephine opening is made at this point and a needle introduced in the direction of the suspected lesion until pus or the resistance of the abscess wall is encountered. When a frontal abscess is suspected a similar process should be carried out in this region.

When the abscess is not located at the expected site the operator has a choice of two procedures: trephination and exploratory punctures in other parts of the brain or ventriculography. The former is preferable since the abscess may rupture into a ventricle if the cerebrospinal fluid is removed too quickly or the internal pressure relations are otherwise disturbed.

**Treatment** — Although the treatment of cerebral abscess consists fundamentally of surgical drainage, opinion differs considerably as to the best means of accomplishing this. The debated points are (1) The most favorable time for drainage and (2) the question of whether conservative or radical drainage procedures should be employed.

The most conservative form of drainage is that of Dandy, who recommends that the abscess be repeatedly tapped through a small trephine opening and that nothing more be done. The basis of the procedure is Dandy's statement that most abscesses will spontaneously sterilize themselves if their intracapsular tension is relieved. While this may be true of the original abscess, the method utterly ignores the opportunity for reinoculation along the needle tracts with the production of secondary abscesses. Whereas several neurosurgeons have had favorable results with this method in occasional isolated cases, none have had the general success experienced by Dandy. In the opinion of the writer the procedure may well fill a useful role as a preliminary or conditional method of treatment. After localization of the abscess, the first evacuation of pus may be accomplished through a simple tap. Should the abscess refill several times or other signs of secondary abscesses along the needle tract develop, the method should be abandoned for one of the more radical operative procedures.

Another conservative method consists of incision of the abscess and drainage through the use of a small rubber drainage tube (No. 18 catheter). Although the procedure is simple and follows sound surgical principles, serious postoperative complications may easily develop. For instance, the drainage tube will not remain in place in the collapsed abscess cavity unless it is sewed firmly to the dura. If this is done, however, there is a grave possibility that the inner end may punch its way centrally into the expanding ventricle with resultant fatal meningitis. Furthermore, the too sudden and complete collapse of a large cavity about a small rubber

tubing permits of the formation of poorly drained pockets which may become secondary abscesses. Grant pointed out that both objections could be materially lessened by delaying the operation when possible until the fourth to sixth week at which time the walls would be well developed and firm. This procedure has been most used in the past.

A more radical treatment of cerebral abscesses was advocated by King in 1924. In this technique the brain tissue overlying the abscess is removed, the top of the abscess cut away, and the cavity and cerebral defect packed with iodoform gauze. The latter is allowed to remain in place for ten to twelve days by which time the entire cavity is lined by healthy granulations. During wound healing herniation is prevented by repeated lumbar punctures. This method has the advantage of thorough open drainage and secondary pockets are rare. It also does away with rubber drainage tubes and the danger of breaking into the ventricles with resultant meningitis. It has the disadvantage however of being a more serious procedure than the ones previously described. There is also a possibility that the wide scar may tend to produce convulsions although this has not occurred to date in King's extensive series. Radical drainage by this method in the hands of King and others has produced excellent results and in the writer's opinion has proven to be the method of choice. Although the various operative procedures used to drain cerebral abscesses have here been discussed at some length warning must be given that treatment of a cerebral abscess should not be attempted by the surgeon unfamiliar with the technical aspects of brain surgery. Few conditions require more particular skill and judgment than the successful treatment of an abscess of the brain. Properly treated the prognosis for permanent cure should be somewhat better than 70 per cent.



## CHAPTER L

### ACUTE CRANIAL INJURY

ACUTE cranial injuries comprise (1) Fractures of the skull, (2) cerebral concussion and edema, and (3) intracranial hemorrhage

#### FRACTURES OF THE SKULL

*A Simple linear fractures* of the skull, of themselves, are not serious and require no special treatment other than a moderate period of rest

*B Depressed fractures* should be elevated as soon as practical to prevent permanent damage to the compressed brain underlying the fracture

*C Compound fractures* require immediate and special attention. Healing by primary union should be aimed for in all fresh wounds. If the surgeon sees the wound within an hour of the accident the following procedure is recommended: radical "débridement" of all devitalized skin and other tissues, thorough disinfection of the parts including the depths of the wound with 70 per cent alcohol and, when technically possible, reapproximation of the wound edges without drainage. A wound thus treated requires close attention for the first week, at the first evidence of infection it should be opened widely and Dakinized.

**Advantages of Primary Closure**—Primary healing of the initial wound has one very great advantage, *i e*, it allows reexploration to be carried out through it. Let us suppose that four or six days after the accident a subdural hematoma is suspected. If the original wound has been surgically closed without infection, it is a simple matter, under novocain, to make a small trephine opening at the site of the fracture, through which the presence of a clot can be easily detected, and if present, drained. Had the original wound been allowed to close by granulation, the diagnostic and curative trephine, so simple in the presence of a clean field, can only be carried out at great risk of producing meningitis or intracranial abscess.

**Healing by Granulation**—When the patient does not reach the surgeon's hands until more than an hour after the accident, bacterial invasion has probably occurred in the devitalized tissues. In such instances it is best to pack the wound widely open, apply wet dressings, and allow healing to occur by secondary intention. The

hazard of meningitis or cerebral abscess is slight so long as the dura remains intact and the scalp wound is kept widely open and moist.

**Treatment of Serious Compound Fractures**—The treatment of severe compound fractures of the head in which the dura is torn and the brain itself is lacerated is such a complicated problem that the writer feels the assistance of a neurosurgeon should be sought in such instances. In the absence of such help perhaps the wisest course is to pack the wound open and keep the dressings constantly moist with a continuous Dakin's drip. Tendency to herniation is combated by frequent spinal taps. The complications are many and the prognosis grave.

### CEREBRAL CONCUSSION AND EDEMA

The most evident physical sequela of severe cerebral concussion is *edema of the brain*. The mechanism need not be here discussed. Suffice it to say that within a few seconds after a severe trauma the brain begins to swell. This expanding pressure against the unyielding cranial vault accounts in large part for the severe headache, restlessness, delirium and stupor variously accompanying blows upon the head. As the pressure exerted by the expanding brain increases and persists the normal circulation of the blood within the brain is interfered with which if continued results in permanent changes in the cellular elements of the cortex and brain stem.

Cerebral ischemia affecting especially the medulla oblongata caused by the tightly swollen brain is the most common cause of death during the first twelve hours after severe head trauma.

**Treatment** The treatment of severe concussion has gone through several changes within the past few years. With the improvement of neurosurgic technic decompressive operations of various sorts became the vogue. Sufficient data was accumulated, however, to prove definitely that patients who were not decompressed did better than those who were. The policy was then adopted of leaving such cases severely alone. But recently Fay of Philadelphia has recalled attention to the fact that edema of the brain produces ischemia by squeezing out the blood, also that the degree of this cerebral ischemia resulting from the swelling of the brain after trauma can be reduced, at least theoretically, in two ways: (1) By the use of dehydrating measures such as the intravenous injection of 50 per cent glucose solution in doses of 50 to 100 cc every three to six hours; and (2) by frequent drainage of the spinal fluid through lumbar tap—50 to 60 cc every eight hours. Assuming there to be roughly 50 to 60 cc of spinal fluid normally contained in the cranial cerebrospinal spaces, the removal of this fluid will allow that much extra cranial space to be occupied by blood. If this blood be in

circulation many times 60 cc of blood would be brought into active physiologic contact with the tissues during an hour.

By the use of dehydrating agents and spinal drainage Fay claims that he has greatly shortened the period of required hospitalization and has lessened the incidence of such post-traumatic symptoms as headache, vertigo, asthenia, etc. Incidentally the taps also tend to free the cerebrospinal fluid of old blood and other debris which tend to clog the terminal subarachnoid spaces from which the spinal fluid must be absorbed. Failure of this may result in non-obstructive hydrocephalus. Fay states there is no danger of producing herniation of the brain into the foramen magnum as a result of the taps provided these are started soon after the trauma. He admits some need for caution if the taps are begun twelve to fifteen hours later.

### INTRACRANIAL HEMORRHAGE

Acute intracranial hemorrhage following trauma may occur in any one of three forms: (1) *Acute epidural hemorrhage* from the middle meningeal artery; (2) *acute basilar hemorrhage* from a torn vessel, usually a venous sinus at the base of the brain; and (3) *acute intracortical hemorrhage* usually associated with laceration of the brain. The chronic subdural hematoma which manifests itself days or even weeks after the trauma will be discussed independently and later.

**Acute Epidural Hemorrhage**—Acute epidural hemorrhage generally occurs from a torn middle meningeal artery produced by linear fracture over the vault of the calvarium. Several branches of the artery may be torn. These hemorrhages being arterial are always acute and require immediate intervention. When properly treated the prognosis is good.

**Symptomatology**—A typical history is that of a youngster who is hit in the temple while standing at bat by a pitched baseball. He receives a slight concussion and is temporarily knocked out. He quickly recovers from his concussion, regains his senses and returns to play. As time goes on, however, he complains of increasing headache and nausea and then once more becomes dull and develops weakness on the side of the body opposite to his injury. These signs may progress to complete hemiparesis and deep stupor.

*The history of recurrent stupor after a period of lucidity is of extreme diagnostic importance.* It represents the effects of a primary concussion from which the patient temporarily recovered followed by a secondary progressive intracranial pressure effect due to an accumulating blood clot. As the intracranial pressure increases the temperature usually rises rapidly, the pulse and respiratory rates are at first progressively slowed but later if the condition becomes critical may accelerate greatly. Paralysis develops. If the intra

cranial pressure is not relieved by removal of the clot, severe cerebral ischemia will ensue followed by death from hyperthermia or paralysis of the respiratory center

**Diagnosis** — Although strongly indicated in the history, the diagnosis is usually definitely established only by exploratory trephine over the site of the suspected clot

**Treatment** — This consists of evacuation of the clot and ligation of the bleeding vessel. The technical considerations are discussed in a subsequent part of this chapter correlating all the phases in the management of an acutely injured head case

**Acute Basilar Hemorrhage** — Hemorrhage at the base of the brain occurs frequently with fractures through the base of the skull. Although the vessels torn may be either arteries or veins the damage usually involves one of the basilar sinuses, the cavernous, great superficial petrosal or sigmoid. These tend to bleed into one of the adjacent subarachnoid cisternæ and thus fill the cerebrospinal fluid and spaces with gross blood. Hemorrhages of this type are extremely grave. The blood corpuscles follow the subarachnoid system over the cerebral cortex and tend to plug the fine capillary spaces in the depth of the sulci thereby interfering tremendously with absorption of the cerebrospinal fluid. There is thus produced an acute hydrocephalus of the non-obstructive or communicating type. (Refer to Hydrocephalus)

The only hope of recovery in such cases lies in keeping the spinal fluid spaces as well cleaned out of hematogenous debris and as well decompressed as is possible. This is best affected by means of frequent spinal drainage of 40 to 60 cc. every six or eight hours. The prognosis in cases exhibiting heavy gross blood in the spinal fluid is despite all treatment extremely bad. Less severe cases showing only traces of blood may recover.

**Subcortical Hemorrhage** — Hemorrhage into the substance of the brain is usually secondary to lacerating injuries from moving fragments of bone. However one occasionally sees massive hemorrhages associated with an almost explosive rupture of the cortex without signs of local displacement of bone, and at great distances from the site of direct trauma. This is the typical and serious 'contra-coup' injury. At other times grave and even fatal injury to the brain may be evidenced by no more objective findings than a few scattered petechial hemorrhages limited to one lobe of the organ and without any free blood in the ventricles or subarachnoid spaces. The hemorrhage within brain tissue tends to 'point' directly into the subarachnoid spaces or indirectly thereto by way of the ventricles. It ultimately appears in the spinal fluid taps where it is undistinguishable from the bleeding due to torn basilar vessels.

**Diagnosis** — The condition is usually undifferentiable, clinically, from hemorrhage due to a torn sinus at the base of the brain.

**Prognosis** — This is grave in all cases. Curiously enough it would appear from the author's observations that the prognosis is better in cases due to direct injury with compound fracture tearing of the dura and brain laceration than in those due to indirect contra-cou trauma.

**Treatment** — There is no specific treatment for this type of bleeding except to keep the spinal fluid spaces drained as well as possible through frequent lumbar taps.

**The Management of Acute Head Injuries** — Certain general principles should guide the management of acute head injuries.

1 *Wounds of the scalp* especially compound fractures should be converted into clean surgical wounds and closed whenever possible. Such treatment comprises thorough sterilization and accurate closure without drainage whenever this is feasible. The purpose is to keep the wound and adjacent skin free from infection so that cerebral exploration may be safely attempted if signs of intracranial bleeding should subsequently develop.

2 *Cerebral edema* is best treated by "dehydration" and spinal drainage as previously described since experience has demonstrated clearly that operation definitely increases the mortality. This same treatment is also indicated for basilar and subcortical hemorrhage.

3 *Exploratory Trephination* The early use of the exploratory trephine (bilateral if necessary) to determine whether or not an epidural hemorrhage exists is indicated in cases where the clinical diagnosis is uncertain and the patient's condition is getting worse. This is an exceedingly simple safe and precise procedure which should be used far more generally than it is. All cases exhibiting unfavorable progress should have early bilateral exploratory trephines performed.

The author's procedure for the management of severe head injuries is as follows. Unless the patient is severely shocked an immediate roentgen ray of the skull is taken. This requires but a few minutes and often yields valuable information regarding linear and depressed fractures. All scalp wounds are treated in a careful surgical manner in accordance with the principles previously outlined. The patient is then put to bed under special observation. Rectal temperature is taken every hour and the pulse and respiratory rates are charted at fifteen minute intervals. Responses are tested every half hour. In testing these simple questions are addressed to the patient requiring simple answers and he is asked to move the fingers and toes of both sides. When the patient is uncooperative the reactions are tested by pressure upon the supra-orbital nerves and by pricking the palms and soles with a pin. While under observation a lethargic patient should be kept off his back as much as possible and be turned from side to side at intervals,

the jaw should also be held forward to prevent the aspiration of mucus. Fluids are given by rectal drip. Narcotics of all sorts are withheld as they mask the patient's responses and specifically depress the respiratory center. Restlessness is treated by large doses of bromides administered orally or rectally. Cerebral edema is combated by the intravenous administration of 50 per cent glucose solution in doses of 1 cc. per kilo of body weight repeated every four hours and by spinal drainage. The latter is accomplished by lumbar puncture performed with the patient on his side and through the slow removal of 50 to 100 cc. of spinal fluid. The procedure should be started promptly after the patient has been put to bed if his condition appears serious and be repeated at intervals of six to eight hours.

A policy of watchful waiting should be followed so long as the temperature, pulse, respirations and general reactivity of the patient remain within limits compatible with life.

**Ominous Signs** — The following signs of impending decompensation however are ominous: temperature rise to above  $103.5^{\circ}$  F.; progressive increase of pulse rate to 150 per minute or slowing to 36; depression of respiration to 14 or 12 per minute or elevation to 34 or 36; or deterioration of responses so that the patient no longer reacts to the vigorous stimuli previously described. Such changes indicate that the brain is not compensating successfully for the altered pressure conditions within the cranial cavity and that changes are under way which are rapidly approaching a state incompatible with life.

**Causes of Decompensation** — The impending decompensation may be the result of cerebral edema alone or of cerebral edema complicated by hemorrhage. The latter may be in the form of an epidural or subdural blood clot or bleeding into the cerebrospinal spaces which interferes with the normal circulation and absorption of the cerebrospinal fluid producing thereby an acute hydrocephalus of the non-obstructive type or the hemorrhage may be associated with contusion and laceration of the brain substance introducing an element of shock.

**Rationale of Surgical Treatment** — Armed with these possibilities it is important to realize two facts: (1) That of all the possible pathologic conditions that may be contributing to the decompensation there is only one that can be effectively treated by surgical measures namely a blood clot on the surface of the brain either outside or inside the dura and that its recognition and treatment must be prompt. (2) The diagnosis of this condition in contradistinction to the other hopeless possibilities cannot in most cases be made with any certainty by any purely clinical means and therefore must depend upon exploratory trephining. This is particularly true when a patient has been continuously unconscious.

from the moment of injury or has been found in an unconscious state with no accurate information available regarding the accident

**Exploratory Trephine** can be carried out under novocain in a few minutes, without shock or risk. If hemorrhage is not found on one side, a similar exploration should immediately be made upon the other side, for it is well known that in a certain proportion of cases, for reasons not clear, symptoms may occur on the same side of the body as the intracranial hemorrhage

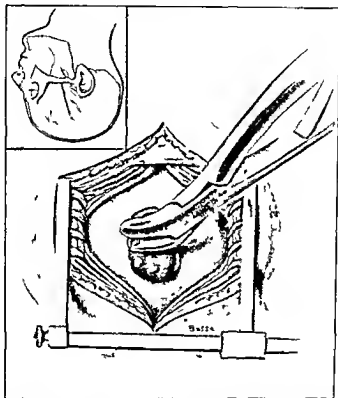


FIG. 279.—Treatment of epidural hemorrhage. Temporal muscle is split as far as the zygoma and retracted to expose the squamous portion of the temporal bone. Trephine opening is made 1 inch above the zygoma. Immediate exposure of the clot occurs. The opening is then quickly enlarged with the rongeur.

**Operative Technic for Trephine Exploration and Removal of Blood Clot**—The operator should be equipped with a good headlight, a suction apparatus with a long slightly angulated tip about one-half the diameter of a pencil, a malleable "ribbon" retractor or spatula an inch wide and 6 to 8 inches long, several long blunt nerve hooks, silver clips such as are used by neurosurgeons for ligating friable arteries in deep wounds, and appropriate bone instruments for getting through the skull.

Infiltration of skin and muscle along the line of incision is the only anesthesia required. The incision is made at right angles to the zygoma, the lower end of the incision resting on the center of this structure (Figs 279, 280 and 281). The temporal muscle is split and retracted, and a trephine opening is made through the temporal bone. If an epidural hemorrhage has occurred from the middle meningeal artery, as soon as this opening through the bone has been

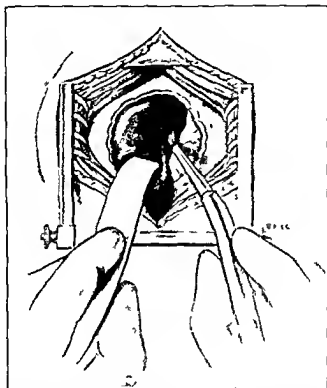


FIG. 280.—The ribbon retractor is introduced through the blood clot as far as it will go toward the apex of the middle fossa and the dura is strongly retracted away from the floor of the skull. The end of the retractor compresses the main branches of the middle meningeal artery and thus controls dural bleeding. The clot is then removed by suction.

made, free and clotted blood will begin to pour through the opening. Should this occur no attempt should be made at this point to control the bleeding, but the opening in the bone should be rapidly enlarged with rongeurs until it will admit the ribbon retractor. This is quickly introduced into the opening, and, without waiting to clear the field of blood, its entering edge is gently, but blindly, advanced between the dura and the floor of the skull as far as it will go toward the apex of the middle fossæ. The retractor is then



lifted up so as to elevate the dura strongly away from the floor of the skull. The tip of the retractor is especially manipulated to bring pressure against the dura.

*Ligation of the Middle Meningeal Artery*—By means of pressure of the retractor against the dura, the main branches of the middle meningeal artery which lie in the membrane directly beneath the retractor can be compressed and the bleeding from them temporarily

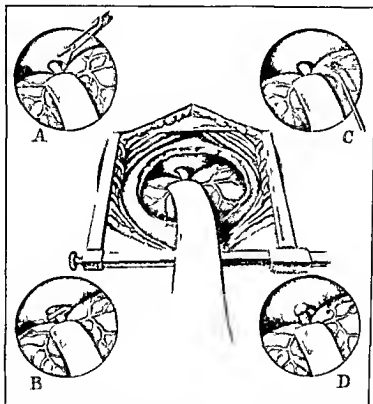


FIG. 281.—The middle meningeal artery is ligated as it emerges from the foramen spinosum and passes to the dura. This may be accomplished by means of a silver clip (A and B) or cotton pellet jammed into the foramen (C and D). A small rubber drain is left *in situ* and the soft parts are closed in layers.

controlled. While the dura is thus being strongly "elevated" with the retractor in the one hand, the middle fossa is cleared of blood by means of the suction tip held in the other. The middle meningeal artery is now brought to light and can be ligated. This is best accomplished with silver clips. If unavailable, a stitch around the artery and through the outer layer (only) of the dura to prevent slipping will be found best. Occasionally it will be necessary to plug the Foramen Spinosum with bone wax or, better still, with a

small spindle of cotton. Blunt nerve hooks are used for this purpose. After the clotted blood has been evacuated and the middle meningeal has been ligated, closure is carried out loosely in layers. It is advisable to leave a good sized rubber drainage tube in place for twenty-four hours.

A common mistake is to look for all the possible bleeding points in the secondary branches of the middle meningeal artery and attempt to ligate these instead of making a single ligature of the main trunk. With a linear fracture extending the length of the skull in an anterior-posterior direction or with a comminuted fracture there may be bleeding points in both frontal and occipital regions which are impossible to reach without extensive and shocking exposure. The essential thing is to ligate the main trunk of the artery before it has branched. Also the operator must be cautioned against wasting time trying to control the hemorrhage during the period when he is getting in. The only effective method of control for such bleeding is by compression of the main trunk of the artery as it courses along within the dura by pressure of the retractor and especially the end of the retractor against it. Since the loss of blood in some cases is considerable it is always advisable to have a donor available.

**Subdural Hemorrhage** — If there is no evidence of epidural hemorrhage when the trephine opening is made the dura should be opened slightly. Subdural hemorrhage however is rare except when associated with cerebral lacerations and subcortical bleeding for which little can be done surgically.

*Surgical decompression per se has no value*

### CHRONIC SUBDURAL HEMATOMA

Chronic subdural hematoma is a delayed manifestation of trauma. Although it occurs characteristically in adults it is encountered sufficiently often in children to merit consideration.

**Pathology** — Chronic subdural hematomata occur whenever one of the small veins leading from the surface of the brain to one of the dural vessels or sinuses is torn. The trauma effecting this is often trivial. The hemorrhage which results may be sufficient to cover the greater part of a hemisphere with a layer of blood 1 to 3 cm. thick in places but the venous pressure is so low that the bleeding stops before sufficient pressure has been exerted upon the brain to kill the patient. This lake of blood between the dura and the arachnoid membrane clots quickly. Almost immediately fibrin released from the surrounding tissue forms a pseudomembrane about it. Into this fibrinous envelope grow fibroblasts and capillaries which rapidly transform it into granulation tissue and ultimately into a thin fibrous tissue capsule with a smooth glistening perioepithelial

lining The process is essentially the same as that by which any foreign body is walled off foreign body in this case being the clot of blood between the dura and the arachnoid

After the capsule has been formed it apparently begins to act as a semipermeable membrane Fluids are drawn by osmosis through the membrane into the capsule but the resultant mixture of diluted blood will not pass outward nor be absorbed As this process of osmosis continues two things happen to the contents of the sac the clot becomes liquefied and increasingly diluted and the capsule containing this mixture progressively enlarges

**Clinical Manifestations**—The initial trauma may be very very slight so that no concussion was ever noted and the original blood clot may have occurred under such slight venous pressure that it produced no pressure symptoms Through osmosis and the liquefaction of the clot a point is ultimately reached where the distended capsule produces symptoms Although this most typically occurs between the tenth and twentieth days symptoms may appear earlier or be delayed for weeks or even months

**Symptomatology**—The symptoms produced by chronic subdural hematoma vary greatly not only in the time of their appearance but in their character They may be focal or general The most common focal symptom is hemiparesis This may however be very slight such as a slight facial weakness suppression of the free associated movements of one arm a scuffing of one foot or the barest suggestion of aphasia The reflexes should of course be correspondingly affected The most constant confirmatory and lateralizing sign is a unilateral dilatation of the pupil which occurs almost always on the side of the hemorrhage

Signs of a generalized increase in intracranial pressure include drowsiness headache depressed pulse and respiratory rates nausea vomiting convulsions and edema of the optic nerve head Ipsilateral signs however occur frequently and should always be kept in mind when a chronic subdural hematoma is suspected A hemorrhage overlying the right hemisphere would then give weakness of the right arm and leg with exaggerated reflexes on the right The explanation for this strange phenomenon is largely speculative but it is probably due to displacement of the brain stem away from the side of the clot with resultant pressure on the opposite side of the brain stem against the sharp edge of the incisural opening in the tentorium It is therefore often impossible to be certain as to which side of the brain the clot is actually overlying

**Treatment**—This consists of a trephine exploration made under novocain over the site of the suspected hematoma The dura is opened through a small incision If a subdural hematoma is present a pale chocolate covered glistening membrane of connective tissue will be found just beneath and adherent to the dura This

is opened through an incision about 1 cm long and the dark syrup-like contents of the sac evacuated. When the washings have become clear, experience has proven that nothing further is required. It is not necessary to resuture the dura or employ drainage. The skin is carefully closed in layers with interrupted sutures. Occasionally symptoms return after a day or two and it may be necessary to remove a few stitches and reirrigate the cavity. For this reason the original wound should be carefully sutured so that this secondary step, if necessary, may be carried out through a clean wound.

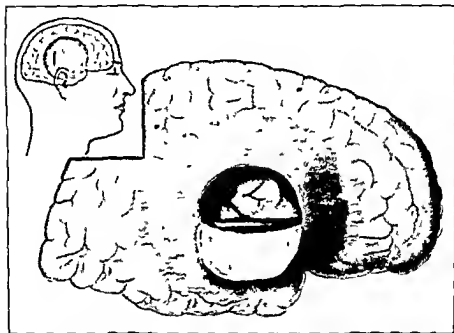


FIG. 282 Chronic subdural hematoma. Drawing of a pathologic specimen showing the fibrous envelope formed about the original clot which has subsequently become liquefied.

The selection of a site for trephining allows considerable latitude, since chronic subdural hematomata tend to spread out over a wide surface of the brain, so that an opening over almost any part of the mido-lateral area will generally reveal the lesion. The writer usually explores through the squamous part of the temporal bone just above the zygoma as for acute epidural hemorrhage. Care should be exercised to avoid injuring the middle meningeal artery as it traverses the dura.

*Bilateral trephination* is frequently necessary and should always be performed if (1) unilateral exploration reveals no clot or (2) when

the patient does not improve after removal of the clot on one side. The first exploration should, of course, be made on the side contralateral to the available (?) focalizing signs, but if this proves negative, trephine should always be immediately performed on the second ipso-lateral side.

In summary the following points should be emphasized:

1 Chronic subdural hematomata may occur where trauma has been negligible, or when the history of trauma is altogether lacking.

2 The symptoms may not make their appearance for days, weeks or even months afterward.

3 Signs are often vague and even when lateralizing signs are present they may be due to a hematoma in the ipso-lateral side.

4 Chronic subdural hematoma must be suspected whenever symptoms and signs of intracranial pressure appear, persist or increase following cranial trauma however slight.

5 The diagnosis of chronic subdural hematoma can usually be made with certainty only after trephine exploration with actual disclosure of the hematoma. The diagnosis can often be disproved only after negative bilateral trephination.

## CHAPTER LI

### ACUTE SPINAL INJURIES \*

ONE of the first facts which emerges from a study of spinal injury is that there exists no constant quantitative relationship between the extent of the injury to the bones of the spinal column and the extent of the injury to the spinal cord. It immediately becomes evident that the neurologic signs following serious spinal trauma are the result of four different pathologic factors acting individually or in various combinations:

- 1 Laceration and crushing of nervous tissue by moving bony fragments at the time of original trauma
- 2 Hemorrhage into the cord
- 3 Edema of the cord
- 4 Continuing pressure upon the cord caused by residual bony deformity

The writer feels that the inclusive term *spinal injury* should be more generally used in referring to this type of case rather than specific terms such as *fractured spine*, *hematomyelia*, or *dislocation*—since none of these specific conditions is apt to occur alone where there has been spinal trauma.

The role of each of the above pathologic factors in the production of neurologic symptoms following spinal trauma will be considered one at a time and on the basis of these considerations suggestions for therapy will be made.

**Laceration and Crushing of Nervous Tissue by Moving Bony Fragments at Time of Original Trauma**—Crushing or laceration by moving fragments of bone at the time of initial trauma is of course the most serious of all the four factors since injuries of this type can never be repaired in any degree either by natural processes or through surgical help.

It would be desirable from the standpoint of prognosis even though it would have little effect upon therapy to determine if possible the extent to which the neurologic symptoms in any given case were due to total section of nerves. This is impossible in the early states of the injury and the writer will not take up space in speculation upon this point.

**Hemorrhage Into the Cord (Hematomyelia)**—Hemorrhage into the cord is an almost constant pathologic sequel of spinal trauma. It

\* Excerpted in part from the author's article in *New York State Journal of Medicine*, vol. 35, No. 5.

is present, in greater or lesser degree, whenever the cord has been contused by a fractured vertebra, and is an important factor in

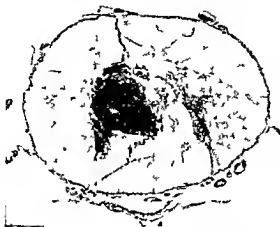


FIG. 283.—In hematomyelia the bulk of the hemorrhage is usually slight. It produces symptoms not by pressure effects on the passing tracts, but by interference with cells and association pathways in the central gray matter of the cord, chiefly the anterior horns. Operation accomplishes nothing in this condition.

producing the symptoms which follow. It may even occur after indirect trauma to the head or body when there are no signs what



FIG. 281.—Atrophy of the intrinsic muscles of the hand occurs when the anterior horn cells are destroyed by hemorrhage. This atrophy causes a typical deformity—the so-called spinal hand.

ever of a fractured spine, and, indeed, sometimes when there is hardly even an adequate history of trauma.





The reason for this predilection of the hemorrhage for the gray matter in the cord is not clear unless it be that this part of the cord is relatively less dense than the peripherally placed longitudinal fasciculi but whatever the reason for it that pathologic fact explains the clinical observation made many times namely that in patients with spinal injuries in the cervical region the paralyses of the legs and bladder may clear up quickly and almost completely while those in the arms and the hands tend to improve much more slowly and much less completely. In addition there usually appears in cases of hemorrhage into the cervical cord a marked atrophy of the muscles of the forearms and hands—such as follows destruction of the anterior horn cells in the gray matter producing the typical deformity seen in cases of syringomyelia or central tumor of the cervical cord the so-called spinal hand (Fig 284)

How shall hemorrhage into the cord be treated? In the writer's opinion not by surgery. For by the time the injured patient has been brought to the hospital has been examined roentgen-rayed and prepared for operation at least one to two hours will have elapsed. By this time the bleeding will most surely have stopped of its own accord since the hemorrhages when seen at post-mortem are never very large. But during this interval more than enough time will have elapsed for a hemorrhage starting let us say in the cervical region (which is the most common site) to have already dissected up and down the cord a considerable distance and to have pithed the gray matter supplying the brachial plexus. Surgery can retrieve nothing here.

It has been urged by some that in these cases the cord should be exposed by laminectomy and attempts made to aspirate the blood within the cord by means of a needle and syringe although it is probable that long before exposure of the cord could be made most of the extravasated blood would have been clotted and would not pass through a needle. Under these circumstances it is then proposed that the cord be split down the dorsal mid line and the clot extracted or allowed to extricate itself. These suggestions however ignore the essential pathologic fact that the hemorrhage produces its chief effect on the cord by invading and disrupting and partially destroying the delicate individual cells and association pathways within the gray matter. Passing a knife blade through a spinal cord in this state will not repair the damage but probably add to it.

What then can one accomplish for these patients? The key to the answer lies in the hope—which is very often a fact—that the destruction of tissues is not as great as would appear at first. In a relatively large number of patients this is true. The return of function is usually not complete although it may be nearly so. This is

particularly true of the legs while the hands for reasons explained above lag behind.

The important points in the treatment of hematomyelia are these:

1 *Prevention of Bed Sores*—This is accomplished by the use of air mattresses and meticulous nursing care.

2 *Prevention of Urinary Infection*—This is best secured by the excellent method of tidal drainage recently described by Munro. Repeated catheterization and the use of the ordinary retention catheter are to be avoided whenever possible in all cases not previously infected since they invariably result in cystitis and other undesirable changes in the bladder. Establishment of an automatic overflow bladder in the absence of infection is usually preferable to catheterization. Emptying can be aided materially by pressure applied at periodic intervals over the bladder (Credé). If the urine is already infected when the patient is first seen free drainage is of course necessary.

3 *Physiotherapy* during the period before return of spontaneous movement—passive motion, massage and heat—to maintain circulation and nutrition and avoid fixation of moving parts.

4 *Systematic exercises* after spontaneous movement has appeared. These exercises should be graded according to function and must occupy the greater part of the patient's waking day.

The end results of conservative treatment for hemorrhage into the spinal cord if these rules be strictly followed are gratifying. In a series of 20 cases of total tetraplegia presumably due to hematomyelia 10 died within a period of a few hours to a few days largely from pneumonia. Of the 10 cases surviving the initial period of trauma 8 showed a very large return of function approaching normal in the legs but less in the hands. Two patients showed no improvement.

*Edema of the Cord*—Edema of the cord occurs in every spinal injury which is severe enough to produce neurologic symptoms. In the acute initial stages immediately following the accident it is a very important factor in the production of symptoms. It is the most frequent and usually the most important cause of manometric block in the spinal fluid during the first forty-eight hours following trauma, a point not sufficiently appreciated. It constitutes a great threat to the life of the patient when the spinal injury occurs in the upper cervical region. Yet it is a subject to which very little attention has been directed.

From the pathologic standpoint the important fact is that the spinal cord is capable of quickly swelling to about twice its normal size within a very few minutes after being traumatically injured. In this state it completely fills the dural sac and distends it under considerable pressure. The microscopic appearance of such a distended cord is contrasted with that of a normal cord in Figs. 286 and 287.

From the *clinical* standpoint the important point to be determined is whether in the presence of such a swollen edematous cord the dura should be opened widely as a decompressive measure or should be left closed. On this point there exist two widely divergent opinions. The writer feels however that both reason and experience counsel the conservative course. Theoretically with the cord so edematous and tense there would be great tendency to sudden herniation and spontaneous rupture if any opening whatever were made in the limiting membrane of the cord represented here by the dura (Fig 288)



FIG 286

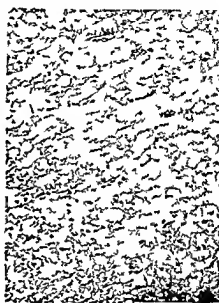


FIG 287

FIGS 286 and 287 The spinal cord after trauma may quickly swell to a most twice its normal size as a result of edema. The above microphotographs are cross sections of the white matter in a normal cord (Fig 286) and a traumatized edematous cord (Fig 287).

That this *does* actually occur when the dura is opened under such circumstances was pointed out a good many years ago by Taylor who reported 2 cases in his own experience in which this had happened. In each instance the patient although temporarily paralyzed at the time he was taken to the operating room for laminectomy was comfortable and excepting for the paralysis in good general condition. In each patient when the dura was opened there was an instantaneous and violent extrusion of the cord through the incision. In 1 case fully 2 inches of the cord was thus blown out within a few seconds. Both patients immediately developed hyperthermia and shortly afterward died. It has been

this writer's fortune while an onlooker at two different operations to observe the same phenomenon. Two other quite similar cases have been reported to the writer in detail by another physician.

It would seem that we are considerably more advanced in the understanding and treatment of *cerebral* edema than in the understanding and treatment of *spinal* edema. We have learned, for example, that the great majority of *cerebral* concussions do better if treated conservatively than if decompressed surgically. Instead of operating we now combat *cerebral* edema with physiologic rest and *dehydrating* agents. The same principles of therapy, the writer feels, should apply to the treatment of *spinal* edema. Surgical decompression is accordingly advised against and the intra-

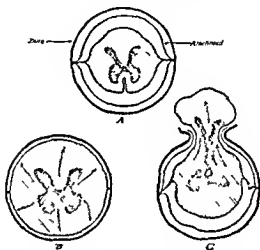


FIG. 288 — A spinal cord—edematous and swollen as a result of trauma—will fill the dural sac and exert great expanding pressure in all directions against its walls. Incision of the dura in such cases is likely to cause instant herniation of the cord with permanent loss of substance.

venous injection of 50 per cent glucose (50 to 75 cc.) is recommended every three to six hours during the first three days.

**Continuing Pressure Upon the Cord Caused by Residual Bony Deformity**—In almost all fractures of the spine there remains some degree of bony deformity, which, if gross enough, may exert continued pressure upon the cord and be in part responsible for neurologic symptoms.

The importance of this residual bony deformity in producing or prolonging neurologic symptoms is, however, in the opinion of the writer, far less than is usually assigned to it. This opinion is based partly upon the anatomic fact that the diameter of the spinal cord is normally only about one-half (in places one-third) of the diameter

of the bony canal so that approximately 50 per cent encroachment upon the lumen of the canal is necessary before serious compression of the cord begins (Fig 289)

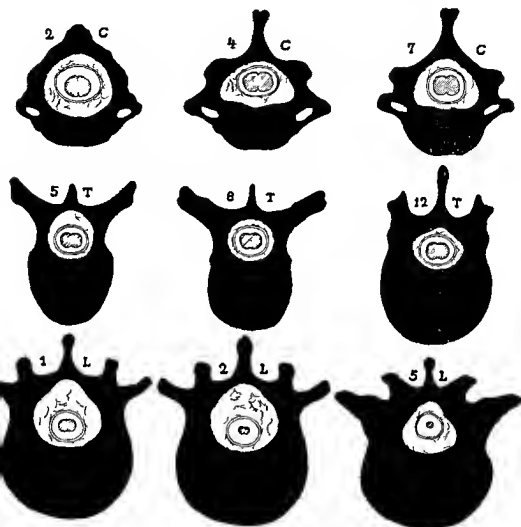


FIG 289 The diameter of the spinal cord is normally only about one-half the diameter of the neural canal so that a 50 per cent encroachment upon the bony canal may take place before compression of the cord begins (The figures here have been traced directly from cross-sections of a frozen calaver)

Even after this degree of encroachment has been reached the cord has considerable capacity for accommodating itself to a change in shape. This has been clinically demonstrated many times in cases of Pott's disease of the spine where extreme deformity frequently occurs without any neurologic signs. Even in our series of acute spinal injuries there is ample proof that considerable encroachment

upon the lumen of the bony canal may take place without causing paralysis.

Correction of deformity is generally desirable however in all cases of fractured spine regardless of the degree of cord compression resulting from the deformity for the reason that any deformity if uncorrected tends to become progressively worse and so may eventually produce greater symptoms.

Deformities tend to follow rather fixed patterns determined more or less by the site of the injury and the initial force of the trauma. A resume of the typical spinal fractures with their resultant deformities is given below together with brief discussion of the rationale and methods for correcting each type of deformity.

Satisfactory reduction is possible in almost all instances by closed nonoperative procedures. In the opinion of the writer correction of the deformity by operative measures is indicated in only the rarest instances. In most cases surgery is not only ineffective but definitely harmful.

After correction of the bony deformity the treatment of a fractured spine is the same as the treatment of hematomyelia or edema of the cord as previously outlined.

1 *Comminuted Fracture of the Lamina*—This type of fracture is exceedingly rare. It is produced by the direct force of a heavy instrument directly against the spines of the vertebra. This is one of the very few fractures of the spine where the writer believes that open operation is definitely indicated. Laminectomy should be performed as soon as possible and all bony fragments removed.

2 *Fracture Dislocation of the Cervical Spine*—A very special function of the cervical spine is mobility. The head must not only move forward and backward to right and to left but also must rotate around the long axis of the body and for this reason all facets along the cervical vertebrae are shallow and all articulations are extremely loose. These anatomic and functional facts predispose to typical fracture dislocation of the cervical spine which is by all odds the most common fracture encountered. In these cases a sudden throwing of the head forward puts extreme stress upon the relatively weak articular processes of the spine one or both of which at any particular level may be broken off. When the articular process on one side only is broken a slight forward rotation of the upper part of the spine takes place when the articular processes on both sides of the vertebra are broken there is a general slipping forward of that part of the cervical spine about the level of the fractures. (Fig. 290.)

Reduction of fracture dislocations of the cervical spine is best accomplished in the writer's opinion by non-operative measures based upon the well acknowledged fact that sustained traction upon the head along the longitudinal axis of the spine will evenly and



FIG. 290 —Fracture dislocations of the cervical spine as above described are by all odds the most frequent spinal fractures. These usually involve the fifth or sixth cervical vertebra—as shown in the roentgen rays ( 807 )

quickly overcome the spasm of the muscles of the neck permitting the falsely locked bony parts to be disengaged and allowing almost perfect realignment (Fig 292)

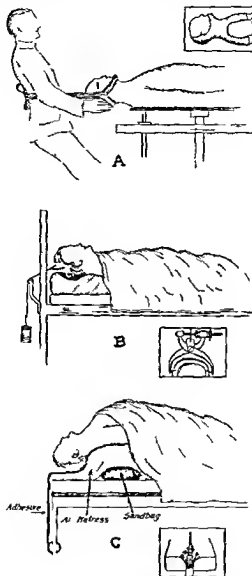


FIG 291 Fracture of locations of the cervical spine are best reduced by non-operative measures. Three satisfactory methods of reduction are here pictured. A Taylor method B method of Crutchfield C Stookey method

Several methods of applying this principle have been devised. Dr Alfred Taylor many years ago advocated the reduction of



deformity by "halter traction" and immediate immobilization in a plaster cast extending from beneath the chin and occiput down over the shoulders and chest (Fig 291 A) We have employed this method many times with most satisfactory results

Another ingenious method for treating these fractures has been proposed by Crutchfield, who devised a set of tongs—resembling ice-tongs—which are applied directly to the bones of the skull and by means of which true skeletal traction is exerted in reducing the muscle spasm in the neck (Fig 291 B) The writer's

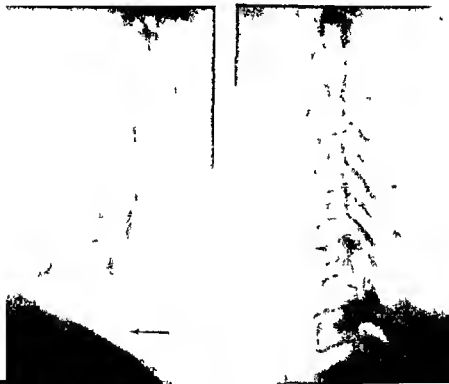


FIG 292—Roentgen rays of a typical fracture dislocation of the cervical spine reduced by the method of Taylor

experience with this method is too limited and brief to justify making a comparison between this method and the earlier method of Taylor. It is true that the "tongs" are much easier to apply than the plaster cast, and they are more comfortable for the patient, but whether the lack of fixation is "desirable" or "safe" has not been settled to the writer's complete satisfaction. In any event, however, Crutchfield has made a distinct contribution to this subject.

A third method for reducing cervical fractures was evolved by

Stoakes at Bellevue Hospital several years ago in which work the writer had the pleasure of assisting him. The essential feature of the method is an air mattress at the head end of which a deep trough is made by means of a broad band of adhesive tape which is fastened to the upper surface of the air mattress, carried over the end of the mattress, pulled strongly downward and fastened to the frame of the bed (Fig. 291 C). The head and neck rest in this trough in a position of acute hyperextension, maintained by



FIG. 291. Compression fractures most frequently involve the bodies of the twelfth thoracic or the first lumbar vertebrae.

the weight of the head itself. Although quite simple, this method has proven very effective, especially in the simpler fractures.

Laminectomy not only is unnecessary in these cases, as pointed out in the above paragraphs, but may even be dangerous. By further weakening the ligamentous and muscular support of the body spine, laminectomy greatly increases false mobility, with added danger to the cord.

**3. Compression Fractures.**—Although compression fractures may occur at any level of the spine, by far the greatest number affect

the twelfth thoracic or the first lumbar vertebra. There is an anatomic reason for this. In the lumbar spine, in contrast to the cervical spine, great mobility is undesirable and strength to support the weight of the body is the prime objective. As a result the articular processes are strong and heavy and the articular facets are deeply placed so that fracture dislocation such as takes place

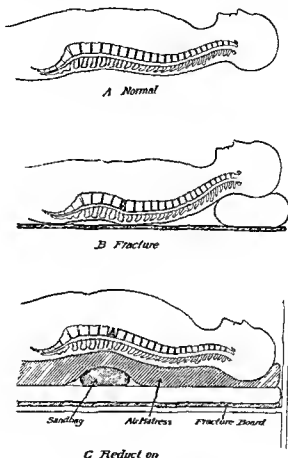


FIG. 231.—Hyperextension of the spine is the most rational and effective means of correcting the bony deformity resulting from compression fracture of the body of a spinal vertebrae.

in the cervical spine is practically impossible. In the cervical spine motion is limited to a slight forward and backward rocking of one vertebra upon another. Attached to these limited joints from above is the rigid and heavy thoracic cage. Any sudden force which throws this structure violently forward tends to force the lumbar articulations past their normal limits of motion. In the neck this would result in a fracture of the light articular pro-

Unfortunately there is little to be done except to make the patient comfortable.

**Delayed Secondary Changes in Spine Following Trauma** In some cases degenerative changes in the bones of the spine are not present immediately after injury or during first hospitalization but subsequently appear weeks or months later. The nature of these changes or the mechanism involved is not clear. They may be upon the basis of destructive arthritis or of thrombosis of nutrient arteries supplying the bones.

Mention of this fact is made however because of the possibility of legal complications arising out of such late change against which proper precautions should always be taken.

for the immediate time being that the interruption in function is due either to edema of the nerve trunk or hemorrhage into it rather than to division of the nerve. With few exceptions such conditions should receive conservative treatment consisting of (1) physiologic rest of the injured part until the post traumatic inflammatory state has subsided (2) local application of heat to the region of the injury to increase the volume of blood supply to the part and thereby hasten absorption of the edema and hemorrhage (3) the later use of passive motion of the paralyzed joint and massage of the involved muscles and (4) electrical stimulation two or three times weekly to cause contractions of the paralyzed muscles until voluntary movements can be carried out. This is to keep the contractile elements alive.

The length of time required for the return of function in this type of injury varies considerably with the severity of the original trauma. In some instances improvement in sensory or motor function appears within a few days whereas in others weeks or months may elapse before there is any evidence of functional restoration. Failure of function to return in cases of external trauma over nerve trunks without actual laceration of the skin may be due to several causes. Actual division of the nerve may result from the causative blow or by moving fragments of bone in fractures. Another cause is the development of scar tissue in the extravasated blood clot about the nerve.

Exploration of the nerve trunk at the site of injury is indicated in general when satisfactory restoration of function does not occur after a suitable period of treatment. Lysis of scar tissue removal of callus or suture of the divided nerve is carried out as indicated at the time of operation.

**Late Effect of Trauma.** At times the paralysis of nerves following trauma to the extremities first appears weeks or even months after the injury. This is especially true of radial nerve injuries associated with fracture of the humeral shaft and of the ulnar nerve following supracondylar fracture. In most instances the paralysis is due to redundant callus incident to repair. In other cases nerve pressure results from scar tissue contraction in the affected region.

Exploration with correction of the causative pathology is indicated in all cases of delayed palsies of peripheral nerves. The exact time elected for operation will depend in each case upon the circumstances attending the injury and the progress of the case under conservative therapy. In instances of radial nerve palsy following severe trauma to the arm exploration of the nerve should be carried out much sooner in cases of fracture than in those without osseous damage.

**Division of Nerves.**—The most important surgical factor in the treatment of a severed peripheral nerve is its recognition and

immediate suture at the time the wound is first seen. At no period thereafter will the opportunity be as favorable for anatomic union and functional recovery. The surgical principles governing the treatment of a lacerated wound with division of a peripheral nerve are few and simple. Thorough cleansing of the part is of prime importance. This comprises debridement and thorough sterilization of the wound and surrounding area with alcohol. The wound should then be carefully explored and the nerve suture performed as meticulously as if the procedure were an elective one carried out in the major operating room. Tension on the suture line should be removed as far as possible. In cases where the nerve has been cleanly cut by a sharp instrument this offers no problem. In others however where bits of ragged nerve tissue must be excised from the two cut ends the resulting defect can usually be overcome by pulling upon the nerve and stretching it slightly. When there are defects of the ulnar nerve at the elbow the two ends can generally be approximated without tension by transposing the nerve ante-

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